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THE EXPRESSION OF THE FACE AND MAN'S TYPE OF BODY AS INDICATORS OF HIS CHARACTER.*

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The muscles of expression could perhaps be more properly called the muscles of emotion. As we all know, if we come down to breakfast with a smile or a frown, we can set the type of our emotions for the day or longer, and make or break the day as far as happiness and often as far as constructive accomplishment are concerned.

The face at first was a primitive affair. It was simply a trap for catching food and, from the first, was very efficient, especially when the lower jaw, which is the only movable part of the face, was added.

All animals except the very lowest have a face. In man, it is not always a success esthetically, being at times only a caricature of a face. No animal takes the liberties with his face that man does, especially the female of the species To

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*This paper under the title "The Muscles of Expression," was used for years as the closing talk of my *Post Graduate Courses* and so to the writer it has a certain sentimental value It was given as a lantern slide talk which permitted it to be personal at times and a bit of this atmosphere has been retained. From time to time the paper has been brought more nearly up to date

All the illustrations are from lantern slides The writer is embarrassed because he cannot readily find references for all the material which has been used He hopes this may be pardoned as the paper has no commercial value except for the engravers who have just turned the lantern slides into cuts for printing

Editor's Note This ms. received in Laryngoscope Office and accepted for publication Oct 30, 1950 developed from his legs but from the gill arches which the fetus of man at one stage of the embryo has in common with fishes.

When the first fishes were formed the seas swarmed with all kinds of invertebrates—sponges, corals, and crabs; and all the major problems of life, feeding, locomotion, and reproduction — sexual and the tamer asexual form — had already been solved for millions of years.

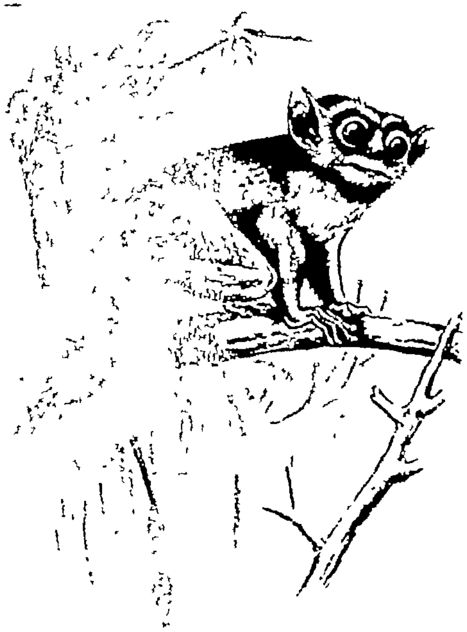


Fig. No. 2.
The Spectral Tarsier of Borneo.
This hideous little creature is supposed by some authors to be an early ancestor of the human race It lived over fifty million years ago

(After H C Raven) (Reproduced from Gregory.)

It was literally a red letter day in evolution when an air-breathing lady-fish (probably a lung fish) didn't get home in time for lunch and laid her eggs on land instead of in the water. This at once set the fashion for other lady fishes of her set and began the land development of vertebrates and soon—that is, in a few millions of years—it led to the next step upward—the development of reptiles.

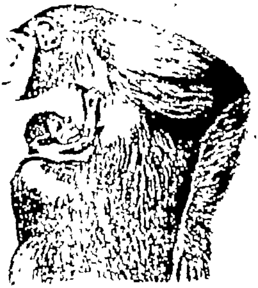


Fig. No. 3.
One of Our Nearest Living Relatives.
Female Chimpanzee and Young.
(From "Almost Human," R. M. Yerkes, Century Co.)
(Reproduced from Gregory.)

At first the land was dominated by reptiles of enormous size and hideous form. Our direct ancestors were small apes who found trees the only safe place to live as the ground swarmed with monsters which, to them, were living mountains. Once up a tree, smell, which up to this time was the most useful sense, became less important as compared with sight. In order to live comfortably in trees, the apes, large and small, had to see where they were going. A jump in the dark, so to speak, could be fatal. Therefore, the eyes of these animals moved from the side of the head, where each eye saw separately, to the front of the skull where the eyes saw as one and gained the ability of measuring distance — stereoscopic vision.

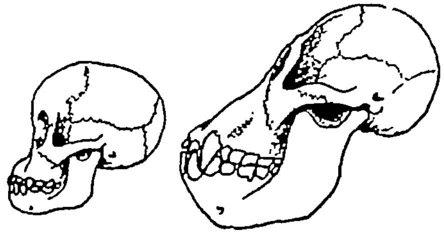


Fig. No. 4.

Profiles of a young and an adult orang-outang showing that the young ape resembles the human more than when it is full grown. (After Wiedersheim.)

(From The Human Skeleton, Walter, Macmillan Co., 1925.)

As the animals increased in size, the long snouts were in the way in climbing, and the angle of the head on the spine was wrong. Accordingly, after a while,—that is, during the customary millions of years—the snout was eliminated, and the head was bent forward to bring the eyes into better position for climbing. By the time all this happened, the trees swarmed with apes and monkeys. Something then happened in the family life in the trees (probably a family row similar to those of today), and some of the apes took to the ground. Here they could stand up on their hind legs and soon learned to walk erect. They discovered that their forelegs were of more use as arms than as legs. Probably the first thing they did with their arms was to throw stones, and soon had the satisfaction of hitting an enemy from a distance and often avoiding hand to hand conflicts. This must have been one of the greatest joys that ever came into animal life at any period. Throwing stones remains a fundamental instinct of boy and man, even today. Further, instead of all the teeth being fitted for grasping and tearing, the back teeth became flat and were miniature millstones, for grinding. Up to the time of this change, all animals had lived by flesh alone. From now on, they could live on grasses and fruits. Some chose only this type of food, and do so to this day. Man is often said to be half savage. Certainly his teeth are: his front teeth still being used for tearing and biting, whereas his back teeth are used for grinding. It is thought that we come from the non-fighting, peace-loving, plant-feeding ape. In this respect, man harks back to the slow-chewing cow-like type. Even today, there are many human cows.

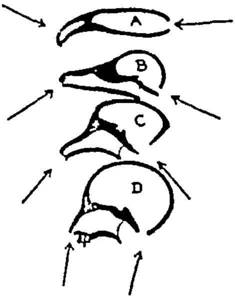


Fig. No 5.

Sagittal sections through the skulls of a salamander (A), deer (B), baboon (C) and man (D).

The arrows indicate an evolutionary change in the relation of the snout and the nasal opening to the foramen magnum. (After Widersheim)

The snout has gradually been eliminated. (From Walter.)

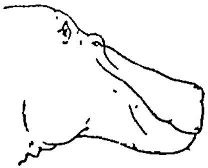


Fig No. 6.

Head of a hippopotamus showing the projecting face. (After Hilzheimer.)
(From Walter.)

Permit me, please, a word or two more to bring evolution up to date. At first, it was held that evolution advanced by constant small changes, but today the belief is that it advanced by jumps; sports, they are called in horticulture; in animals and man they are called mutations. The atomic physicists say that all energy including the energy back of evolution moves in jerks like the hands of a watch. These jerks are now called waves. Thus, the quantum theory (quantum meaning a discrete bundle of energy) was developed and this theory plus the theory of relativity rules the world of physics today. As things are at present, it is easy to admit that this is a jerky world.

Still speaking of evolution, but not so technically, I should like to add the following condensed quotation from Jeans' "The Mysterious Universe" (page 26):

> Cosmic radiation from outer space falls on the earth in large quantities, and its power of destruction are immense. Every second it breaks up about twenty atoms in every cubic inch of our atmosphere, and millions of atoms in our bodies. It has been suggested that this radiation falling on germ plasm may produce the spasmodic biological variations (mutations) which the modern theory of evolution demands. It may have been cosmic radiation that turned monkeys into men." At Bikini all personnel were removed to six miles from the site of the bomb explosion, yet with many nurses their periods stopped for some months, and many men lost interest in their usual dates.

The following has been given as the future evolutionary changes due to come in man: As we are progressively losing our teeth, our faces are shriveling while our brains are growing larger and more powerful. Owing to our erect position, we are losing the lumbar curve of our backs, and the lumbar and sacral vertebrae as well as parts of the pelvis are becoming fused, which means that Caesarean section will, in time, become the normal method of childbirth. Language will be so developed and people will think such advanced thoughts that many will not be able to understand each other, and the weather then will not be the chief topic of conversation as it is today with most of us.

THE MISSING LINK.

Even before Darwin's day (1859—"The Origin of the Species"), there has been a vigorous hunt for the "missing link."

In 1824 (126 years ago) in South Africa, a short distance from the Kimberly diamond mines, the crucial parts of the prehistoric skeleton of a six-year old child were found. The parts were a skull and a number of teeth and they had enough human characteristics for this child to have the honor of being the nearest to the missing link yet found. It is called "Dart's Child," after its discoverer.

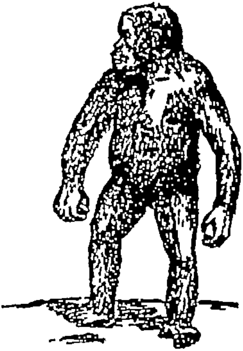


Fig. No. 7.

Hypothetical Sketch of "Dart's Child," the Southern Ape of South Africa (Source mislaid.)

THE SHARK'S FACE AND OURS.

The shark's face shows us our facial anatomy reduced to its lowest terms. We are nearer the shark than any other animal. The skull of the shark has the same twenty-eight bones that are in the skull of man. In the face of the shark, man can see his own face, as in a glass, darkly. Classes in biology the world over use a relative of the shark, the dogfish, as an epitome of the ground plan of human anatomy. This is taken as an insult by the fundamentalists, but such are the facts of comparative anatomy.

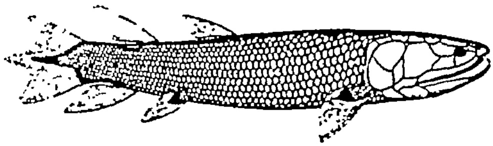


Fig. No. 8.

Devonian "Lobe-fin," first claimant in the line of the ancestry of the higher vertebrates.

(After Bryant.) (From Gregory.)

In certain fishes, the temporal muscle had so much work to do that it tore a hole in the side of the skull. This hole is filled up in the skull of a man, but a depression remains where the hole used to be. This is the temporal fossa and is one of the most conspicuous markings on our skulls.

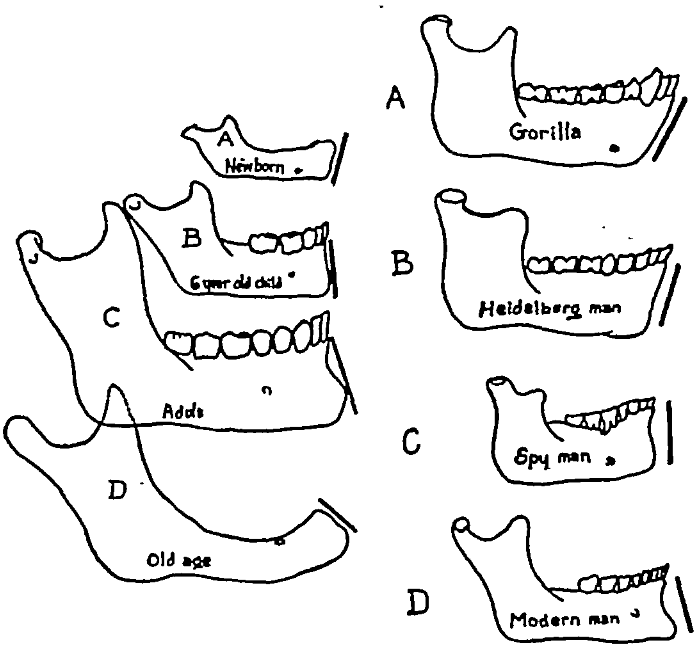


Fig. No. 9.

Changes in the chin-angle during the life of the individual.

Changes in the chin-angle during the history of the race. Man is the only animal who has a projecting angle of the chin.

(From Walter.)

There was another early change in the form of the skull and, so, of the face. From the rim of the orbit, posteriorly and inferiorly, a bridge of bone was developed. This bridge, the zygoma, ran backward horizontally from the angle of the orbit and from the cheek bone (malar) to join the skull in front of the ear, and ended by making the anterior part of the joint of the jaw. On its way back, it is fully an inch from the side of the skull; that is, the temporal fossa is that deep where the tendon of the temporal muscle runs inside it to be attached to the lower jaw. Without the bridge, there would be a tremendous hollow behind the eye on the side of the head, and every time we closed our jaws the temporal muscles would bulge out like a second pair of ears.

SCIENTIFIC DETECTIVE WORK.

The molar teeth and bits of the lower jaw have given the best clues of the progressive change-over from ape to man. A bit of lower jaw with three teeth gave anthropologists the first clue to the changes. As apes are often larger than men, their heads and teeth, especially their molars, are larger. The cusps of the molars have a regular order and change in a regular manner as man is approached (see Gregory, p. 151).

The plotting of the changes in prehistoric teeth is one of the best pieces of detective work that science has ever done. It was a planned affair, not an accidental discovery like penicillin.

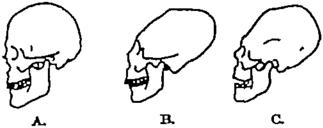


Fig. No. 10.
Artificial Deformities of the Skull.

Fig. A. Occipital deformity of the skull due to cradle-board Southwest U. S. Indian.

Fig. B. Fronto-occipital flattening of the skull. Northwest coast Indian.

Fig. C. Conical deformity due to bandages. Peruvian Indian.

(Am. Museum of Nat. Hist.)

Fig. No. 12.
Woolly Rhinoceros.

THE APE CHARACTERISTICS.

The ape has no chin, a flat forehead, a horizontal ridge of bone over the eyes, and a sagittal crest along the middle line of the top of the skull. The hard palate is wider in front, rather than behind. The mental tubercles for the attachment of the tongue muscles of man are replaced by a fossa. Both the lower jaw and the teeth of the ape are much larger than in man.

In connection with the wanderings of the tribes of prehistoric ape-like men, one has to remember that there were three ice ages, each with an interglacial period when the climate was livable for the ape-like men. They roamed and hunted in the valleys, and on the plains. They skirted the edges of the glaciers, and the Arctic animals came down near enough to be hunted. These included the mammoth, the woolly rhinoceros, and the equally well protected elephant. When the inter-glacial period ended and the ice moved south again (even, at one time, covering all of France), the ape-men took to the caves against the cold. The fossil bones and the stone implements and bone ornaments date the periods of the different races of prehistoric man. As stated above, the first caves to be explored were in Wales; then a few were found in France, and finally, they were found all over that country. A cave forty miles from Pekin, and one of the richest, was half explored when the Second World War stopped the work. Next to caves, limestone deposits which are being worked today for commercial purposes have yielded a wealth of bones of prehistoric animals and prehistoric man. These were regularly burned for lime and many a precious specimen linking apes and man has been lost. One cave which was used as a sepulcher was panelled with the shoulder blades of a mammoth, and the femurs of this animal were piled up as we stack cord wood, and ash heaps showed they were burned as wood, on account of the large amount of fat in the marrow.

Fig. No. 13.

Neanderthal Man.

There were a number of "Gardens of Eden"; that is, caves of ape-like men have been found in India, Java, China, Central Asia and Africa. There were, therefore, an equal number of Adams and Eves.

In the very early days of the earth, Ireland and England were one, at least geographically, and were joined to the continent of Europe. Africa also was joined to Europe by Gibraltar, Italy, Sicily and Malta. Alaska was a part of Siberia. The prehistoric tribes, therefore, could, and did, travel all over the earth by land, even reaching North and South America. Animals which we know today only in the tropics, like the elephant, the two-horned rhinoceros and the lion, got as far as southern Siberia. Both ape-like man and animals anticipated Horace Greely's advice, and went West.

THE ERECT APE MAN OF JAVA.

Tribes of Forest Apes (our first ape ancestors) roamed over France, Spain, Africa, and South Eastern Asia. One of these, the Erect Ape Man of Java, is called the first human being. He had a big brain, 1550 cc. This is 200 cc. larger than the average human brain, which measures 1350 cc. This ape walked erect, could talk, knew fire, and made crude stone implements. He and his race were giants; as to his intelligence, he would be rated today, probably, as a sub moron.

THE PEKIN MAN.

The Pekin Man is called the first cousin of the Java Ape Man. He was found in a cave forty miles above Pekin, China. Placed next in line of success is the Heidleberg Man, whose period is given as the Second Ice Age.

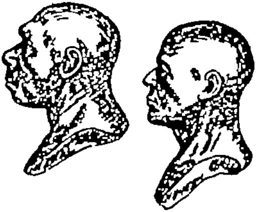


Fig. No. 14.

Neanderthal Man (left) and Cro-Magnon Man.

The Cro-Magnon man is rated as the most perfect physical type of man that ever existed.

THE NEANDERTHAL MAN.

The period of the Neanderthal man is also the second ice age, and he lived in Southern Germany. This was a large tribe which populated western Europe for many thousands of years. Many of his caves have been found. He buried his dead, and seems to have believed in life after death. He used fire and invented pressure chipping. This was a method of making stone knives with edges as sharp as our modern razors. This invention is ranked as of equal importance with the discovery of the crucible for melting iron. The Neanderthal Man disappeared 25,000 to 30,000 years ago.

THE CRO-MAGNON MAN.

The Cro-Magnon man was named from the French town where he was first found. His tribe spread widely over western Europe and became the predominant race, and our nearest ancestor. These men were the finest, physically, the world has

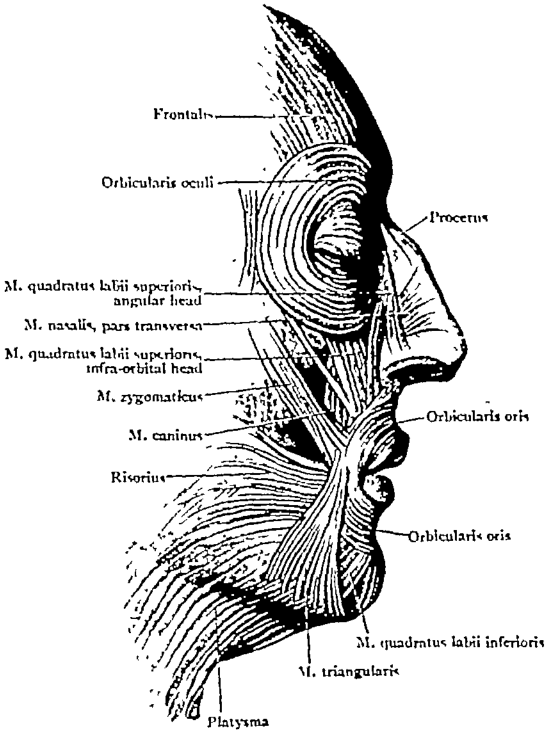

Frontalis
Orbicularis oculi
Procerus
M. quadratus labii superioris, angular head
M. nasalis, pars transversa
M. quadratus labii superioris, infra-orbital head
M. zygomaticus
Orbicularis oris
M. caninus
Risorius
Orbicularis oris
M. quadratus labii inferioris
M. triangularis
Platysma

Fig. No. 15.

In this cut the Zygomaticus Minor muscle is made a part of the Quadrate muscle of the upper lip, and is not given a separate label. It lies to the nasal side of the Zygomaticus Major. In this article the writer considers the Zygomaticus Minor a separate muscle, and a companion muscle to the Zygomaticus Major.

(From Cunningham.)

ever known. They were six feet tall and had big brains. They were all said to be handsome, as we, their descendants all are not, and their cave drawings and paintings were the beginning of modern art, and much better than some of it, and they started the first basic elements of civilization.

THE MUSCLES OF EXPRESSION.

When we examine the face of man, we find that it is composed chiefly of holes,—the nose,—the orbits,—the mouth. These are more or less visible. Five invisible cavities,—the antra and the frontal sinuses and the ethmoidal cells,—complete the hollow shell which makes the upper part of the face. The lower jaw is the only solid and movable part. The orbit is 50 per cent larger than the antrum. The length of the orbital fissure is twice the length of the opening into the nose. The size of the mouth defies comparison.

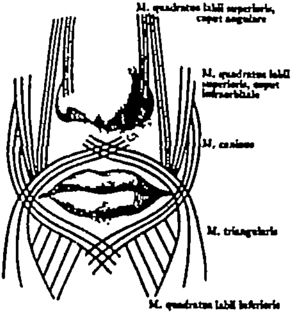

M. quadratus labii superioris, caput angulare
M. quadratus labii superioris, caput infraorbitale
M. caninus
M. triangularis
M. quadratus labii inferioris

Diagram of the Orbicularis Oris Muscle.
The fibres which enter it from the buccinator are not represented.

Fig. No 16.

(From Cunningham.)

The muscles of the face are divided into two groups; the superficial muscles, which are attached to the skin and are called the muscles of expression, and a deep set attached to

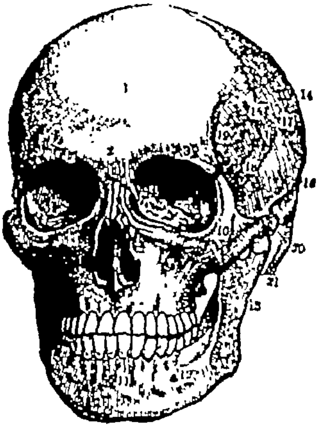


Fig. No. 17

Front View of Skull.

In following the phylogenetic series up to primates and man, a considerable extension of these two layers over the head and face is noticed, and as they meet the eyes, nose, and ear and lips; they give off slips for the regulation of these parts.

THE MUSCLES OF EXPRESSION.

The names of the muscles of expression are so aptly descriptive that simply giving their names, stating a little about their origins and insertions, and reproducing a few anatomical plates so that the muscles can be visualized, the writer considers sufficient for the purposes of this paper.

The muscles of expression bring about the changes of the face which characterize the various emotions, by acting on the forehead, the eyebrows, the corners of the eyes, the corners of the mouth, and the furrow from the side of the nose to the upper lip, known as the naso-labial line.

A sphincter muscle closes both the eyelids and the lips. All the lip muscles take part in the sphincter. The sphincter of the eyelids is divided into two parts, the upper part serving the eyebrow, the lower part the lids.

THE ORBICULARIS PALPEBRARUM.

This is the sphincter muscle of the eyelids. It is divided into two parts. The upper and stronger part lies on the upper margin of the orbit. It extends upward and interlaces with the frontal division of the occipito-frontalis muscle, and mixes also with the corrugator supercilii—a small horizontal muscle just above the eyebrow which, with its mate of the opposite side, makes the two vertical ridges between the eyebrows.

THE ORBICULARIS ORIS.

This is the sphincter of the lips. The greater part is a continuation of the buccinator forward and around both lips; all the muscles of the lips contribute to it.

THE LEVATOR OF THE UPPER LIP AND THE WING OF THE NOSE.

This small muscle has the longest name of all the muscles of the body. It rises from the upper part of the ascending process of the superior maxilla. As it runs downward it divides into two slips, the upper one going to the wing of the nose, while the lower one continues to the upper lip. It is in close relation to the naso-labial fold, and so plays a significant rôle in certain expressions of the face.

THE ZYGOMATICUS MAJOR.

This muscle springs from the malar bone in front of the zygomatic suture. It runs to the corner of the mouth, attaches to the skin, and blends with the sphincter of the lips and the depressor of the angle of the mouth.

THE ZYGOMATICUS MINOR.

This slender but sturdy muscle starts from the malar bone in front of the zygomaticus major and runs to the upper lip. It influences the shape of the naso-labial fold. Modern anatomical nomenclature joins it to the outer part of the levator of the upper lip, and it reaches the upper lip through this. It loses its old name of zygomaticus minor.

THE LEVATOR OF THE UPPER LIP.

This is a flat band of muscle which rises from the lower rim of the orbit and runs to the upper lip.

THE LEVATOR OF THE ANGLE OF THE MOUTH.

This muscle springs from the upper part of the canine fossa of the superior maxilla just below the infra orbital foramen, runs to the angle of the mouth and continues on to the lower lip thus surrounding the angle of the mouth. By contracting it raises the angle of the mouth.

THE DEPRESSOR OF THE LOWER LIP.

This is a strong, quadrate muscle which has a linear origin from the lower edge of the lower jaw starting from the symphysis and ending a little beyond the mental foramen.

THE DEPRESSOR OF THE ANGLE OF THE MOUTH.

This is a flat triangular muscle which rises from the external oblique line of the lower jaw anteriorly. It runs upward to the angle of the mouth and above and around it into the upper lip. It depresses the angle of the mouth.

The muscles of expression are ideally simple, and splendidly efficient. They come from where you would expect, and are aptly named. Two chief muscles are sufficient to control the lower lip, but nature found it harder to control the upper lip and had to devote five muscles to it. It is easier to close the eyes than the mouth. I omit a too obvious comment on this.

THE MUSCLES OF THE NOSE.

The compressor of the nose springs from the superior maxilla close to the bony rim of the anterior nasal opening. The muscle and its mate from the other side join by a medial aponeurosis and cover between them the cartilaginous portion of the nose.

THE LEVATOR OF THE UPPER LIP AND THE WING OF THE NOSE.

This muscle has been described before. It is mentioned again because the upper slip of the muscle goes to the wing of the nose, and so makes it a muscle of both the nose and the upper lip.

THE PYRAMIDALIS.

This muscle springs from the aponeurosis of the compressor of the nose. It runs upward on the nasal bone, some of its fibres reaching the skin of the lower forehead, while others join the frontalis muscle. Through its pull on the compressor of the nose by its origin from the aponeurosis of this muscle it probably has some control of the naso-labial fold; it influences the shape of the eyebrow and the lower part of the forehead.

THE DEPRESSOR OF THE SEPTUM OF THE NOSE.

This muscle springs from the incisor fossa of the superior maxilla. Its outer margin blends with the compressor of the nose. It arches upward and forward and attaches to the lower and back part of both the wing and the septum of the nose.

THE DILATOR MUSCLE OF THE NOSE.

This muscle is composed of two slips. They rise in the dense tissue at the lower and outer part of the nose above the nasal opening. The anterior slip is placed on the lower part of the side of the nose near the tip. The posterior slip lies just behind.

The muscles just described open and narrow the nostrils. The nostrils open wider in sniffing and in deep breathing through the nose. In man and animals the nostrils dilate in sexual excitement. A familiar example is the tossed up head and the quivering and dilated nostrils of the expectant stallion.

In the description of the muscles of expression, especially with the names, the write has mixed up Latin and English. This was done hoping to make the anatomy less formidable.

THE FACE AS AN INDICATOR OF CHARACTER.

Ever since Adam took his first long, loving look at Eve, the face has been considered an expression of character. Roughly this is true, but we are constantly getting fooled. Eve, the apple and the snake fooled Adam. Cain killed Abel, and Cain must have looked like his father, his mother, or God, his only three ancestors,—and they probably had attractive faces. The chain stores won't trust any man's face, although someone has to trust them. Nevertheless, the face, as I have just said, is roughly an expression of character. We have the stolid face of the Indian, the poker face of the gambler, and the kindly face of the motherly old lady or the motherly old man. Of course, too, we have the face of the modern young lady, but this is seldom her own, — and the only safe judgment which can be made about it is that it is often distractingly pretty and fatally alluring.

Speaking of telling a person's character by his face, giving a post graduate course often is no mean way.

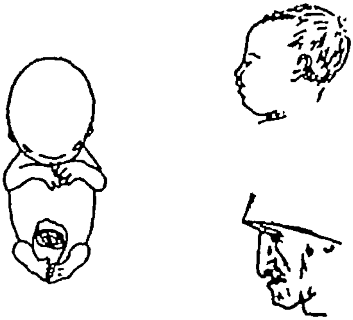
The muscles of the face in expressing emotion act upon the eyelids and lips, but chiefly on the corners of the eyes, and the corners of the mouth; also, they act upon the naso-labial line, the furrow which runs from the side of the nose to the upper lip. These parts (Bell) have the least expression in animals, for they have no eyebrows and no power of elevating or depressing the angle of the mouth.

The most remarkable muscle of the human face, according to Bell, is the corrugator supercilii. It knits the eyebrows and gives the idea of mind behind the face. The next most expressive muscle is the depressor of the angle of the mouth. It is peculiar to mankind and expresses the emotions of contempt disgust and hatred.

In carnivora, the facial muscles run to the angle of the mouth. In herbivora, they run to the lips so that they can be pulled away from the teeth in grazing.

Writings on the subject of the face as an index of character began at least four hundred years ago. To us, fancy, if not foolishness, predominates in them, and they contain many loose observations posing as philosophy.

For the last hundred years, and today, the investigations concerned with this subject have been thoroughly scientific. Darwin wrote on the "Expression of the Emotions in Man and Animals," and the English physiologist, Sir Charles Bell (1844), celebrated for his studies of nervous system, and whom I have just quoted, also analyzed the expressions of the face.



A human embryo at eight and one-half weeks, showing relatively large size of the head. (After Kollmann.)

The profile of infancy, and the profile of old age.

(After Sir Chas. Bell.)

Fig. No. 18.

The Anatomy and Philosophy of Expression. Sir Charles Bell—Fifth Ed. Bohn, London, 1865, p. 45.

EARLY WRITERS.

Astrology. CARDAN. *1501-1576* (448 years ago).

According to the books, geniuses are often illegitimate. The Italian Cardan was in this class, and was the earliest writer on the face as an indicator of a person's character that I have noted. Over four hundred years ago he wrote a book on this subject. In the variety of his attainments, he rivaled Leonardo da Vinci.

As an astrologist, he believed that the stars determine a man's character. When you consider what the cosmic rays are continually doing to us, he may have been a bit on the right track. He held that man's character was shown in his face, especially by the furrows of the brow. In the book which he published, there are illustrations of 700 variations of possible lines on the forehead, each one having a special meaning. To the writer this seems crazy.

Of the other early writers, LeBrun wrote on the resemblance of certain types of the human face to the heads and faces of animals.

Levater wrote a book entitled "The Art of Knowing Man by His Countenance." In it, he gets off on chapters devoted to imaginations, and envies,—warts and beards,—and to lines of animality.

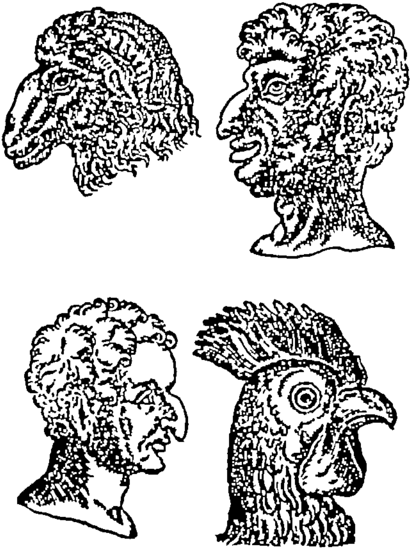


Fig. No. 16.

Le Brun gives many plates showing the resemblance of many human faces to the faces of animals Two of his cuts are shown here. He was especially interested in the relations of the countenance to character.

Sue, in Paris, in 1797 (only 153 years ago), is maudlin in his sentimental approach to the study of physiognomy. For instance, he says: "A mouth delicate and pure is, perhaps, one of the best recommendations." He stops at nothing, and speaks dogmatically of the physiognomy of fishes, serpents, grasshoppers, and intestinal worms. "Intestinal worms have a very decided physiognomy, the character of which inspires in man sorrow and awe." For once, I might agree with him.

From now on, what I have to say, with one exception, is scientific and makes present day sense.

CAMPER. *1722-1789.*

Camper was the first to chart the facial angle. He analyzed the action of the facial muscles, and laid down the general rule that the contraction of each muscle of the face produced in the skin one or more folds, the direction of which is perpendicular to that of the muscle. This is true of almost every muscle of the face. The frontal is the most striking example.

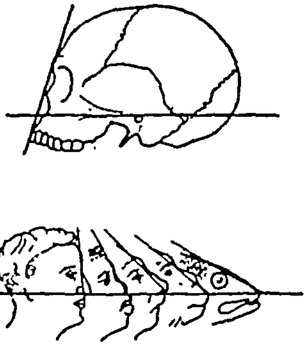


Fig. No. 20.

Upper figure. A profile of a skull showing Camper's angle.

(After Topinard.)

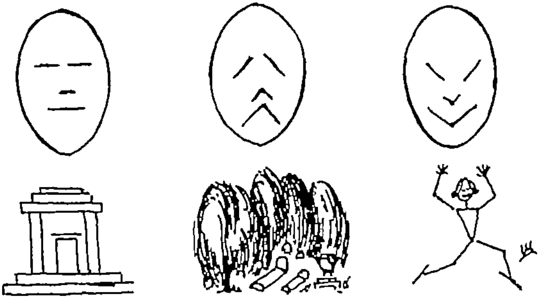
Lower figure. The evolution of the facial angle.

(After Witkowski.) (From Walter.)

SUPERVILLE.

In 1827 (123 years ago), Humbert de Superville published a book entitled, "Des Signes Inconscients de l'Art," which, as best I can translate it, means: "The Unconscious Suggestions of Art." The author gives three drawings of the human face, in which the lines represent the eyes, the lower boundary of the nose, and the lips. In the first drawing, the lines are all horizontal; in the second, they are all inclined downwards and outwards from the median line, and, in the third, all the lines are inclined upwards and outwards. The author states that the first figure (with the lines horizontal) produces an impression of calmness, greatness, and constancy; and he adds that, likewise, in nature and architecture, horizontal lines give rise to the idea of calmness, stability, and grandeur; the cedar, with its horizontal branches, is of all the trees the one that gives this impression in the highest degree. The second figure (with the lines oblique downwards) gives an impression of sadness, pain and grief; and the writer compares the direction of the features of such a countenance with the direction of the architectural lines in tombs and funeral monuments, and to branches of the trees which everywhere are planted, in preference to others, in cemeteries (weeping willows), and whose branches always hang obliquely. Lastly, the third figure (with the lines obliquely upwards) gives rise to the impression of gaiety, levity, inconstancy; and to continue the preceding comparisons, Chinese architecture with its lines oblique and diverging upwards and outwards, can never, at least in the eyes of a European, produce an impression of greatness and majesty.

All the muscles which take part in the expression of pain sadness and contempt help to incline the features obliquely downwards and outwards, some by acting on the line of the eyes, others on that of the mouth.



1. Calmness. 2. Sadness 3 Gaiety—Inconstancy.

Fig No. 21.

This is a rough blackboard drawing by the writer of de Superville's three fundamental expressions of the face.

Having been with the men of my Post Graduate Course for seven weeks, it was easy in this closing lecture to attach appropriate names from the members of the class to each of these figures. These always brought down the house, so to speak, and probaly were the best remembered part of the talk. (From Artistic Anatomy, Duval, pp 281-285-286.)

I said, further back that, with one exception the rest of the paper would be purely scientific according to modern standards. Here is the exception.

PHRENOLOGY.

One of the comparatively modern ways of telling the character of a person is to chart the skull, assigning the various attributes of the mind, body and soul to special bony beds or bumps on the skull. Man's character, according to the cult of phrenology, is summarized on his skull by bumps and depressions. I will agree that bumps have a lot to do with making a man's character. These bumps, the bumps of life, though often hard are not bony.

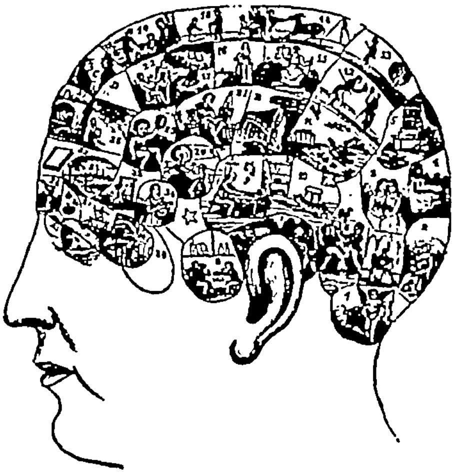


Fig. No. 22.

The Phrenological Chart of the Skull.

Notice figures 1-2-3-4 and 6. (After Fowlers and Wells, Practical Phrenologists and Publishers, 131 Nassau St., N.Y.)

In many cities on the harbor excursion boats phrenologists frequently plied their trade. A passenger paid twenty-five cents to have his head examined and his bumps evaluated. The chart which was given him was always flattering, and it was carried home for the family archives.

The writer was interested to see that, as in the illustration, on the back of the head where amativness is located, Cupid, the marriage ceremony are shown, and two boys in a fist fight. Belligerency comes next to love.

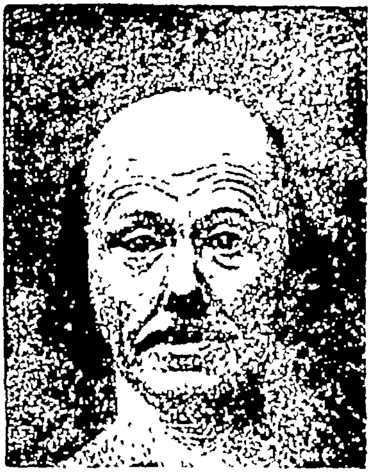
DUCHENNE. *1806-1874.* (144 years ago.)

Darwin wrote "The Origin of the Species" in 1859. Later (1874) he wrote a paper on "The Expression of the Emotions in Man and in Animals." The illustrations of Darwin's paper were mostly reproductions of photographs published by Duchenne ten years before. These had lain neglected until Darwin made use of them. Then due credit was given to Duchenne as the one who had introduced the experimental study of physiognomy.

In 1874, in the course of anatomy in the School of Fine Arts, Paris, the French began to devote several lectures on what must be called the primer or grammar of physiognomy. Happy in seeing his works included in this classical course of instruction, Duchenne, who died a short time later, gave to the School of Fine Arts the complete series of his original photographs, and this beautiful collection is today one of the most valuable in the French museum of anatomy (Museum Hugnier).

Duchenne began his experiments on the head of a criminal executed by the guillotine. He exposed a muscle of the face by dissection so that he could excite it singly by the galvanic current. He had to work fast because muscles retain their excitability for only two hours after death. He photographed the facial expression produced by the contraction of the muscle. Soon he had the good fortune to find an almshouse inmate whose face, from a nerve ailment, was insensitive to pain. His experiments on this man gave the brilliant photographs which Darwin borrowed.

In order to understand the mechanism of the frontal muscle, it is necessary to consider it as taking its fixed insertion at the posterior part of the skull, by the epicranial aponeurosis and the occipital muscle; its free insertion is in the deep



CONTRACTION OF THE FRONTAL MUSCLE
(expressions of *attention* and *astonishment*)

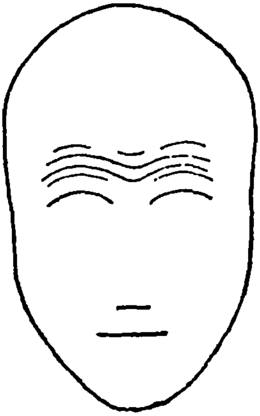


Diagram of the *frontal muscles* (*attention*).

Fig. No. 23.

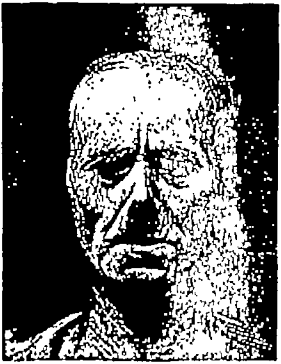
The Muscles of Expression from the Atlas of Duchenne. Artistic Anatomy—Duval, Cassell and Co., London, 1898.

(This plate and the five companion plates which follow are all from Duval.)

surface of the skin of the eyebrow. The frontal, therefore, in contracting, draws this skin from below upwards, and consequently raises the eyebrow, and causes the transverse folds to appear in the skin of the forehead.

In examining a face in which this muscle is contracted, we perceive that it expresses attention; if the contraction of the muscle is very great, this expression of attention changes to that of astonishment.

The superior orbital orbicular muscle (muscle of reflection) is placed beneath the skin of the eyebrow, and its fibres describe an arc with the concavity downwards, of which each extremity is adherent to the corresponding part (internal or external border) of the orbital opening.



THE UPPER PART OF THE ORBICULARIS PALPEBRARUM (*reflection*).

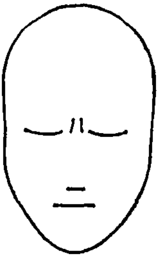


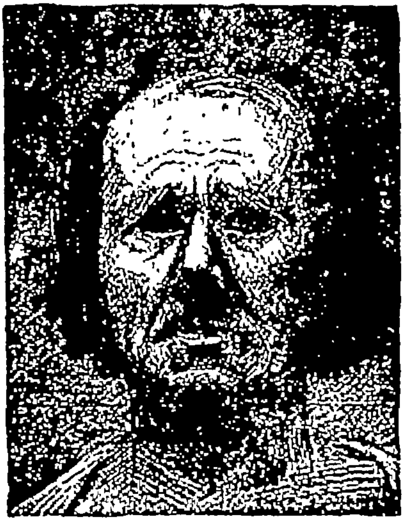
Diagram representation of *reflection, meditation.*

FIG. No. 24.

Figure 24 is the representation of reflection, characterized by the absence of the frontal folds, the depression of the eyebrows and the presence of two small vertical folds in the space between the eyebrows, to which the depression of the eyelid gives rise. This representation of reflection does not acquire its full significance until we compare it with that of attention.

Superciliary muscle (or muscle of pain).—This short muscle is deeply hidden beneath the skin of the region of the head of the eyebrow. Its fixed insertion is into the frontal bone, above the superciliary arch. From this origin, its fibres are directed outwards and slightly downwards to be inserted into the deep surface of the skin at the middle of the eyebrow.

The great zygomatic muscle (or muscle of laughter).—This muscle, called also the external oblique elevator of the commissure of the lips, has its fixed attachment on the cheek



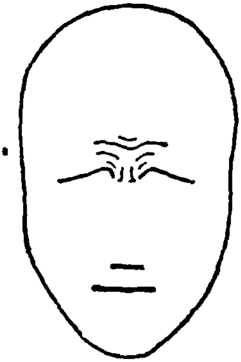


Diagram representation of sorrow.

SUPERCILIARY MUSCLE (*sorrow*).

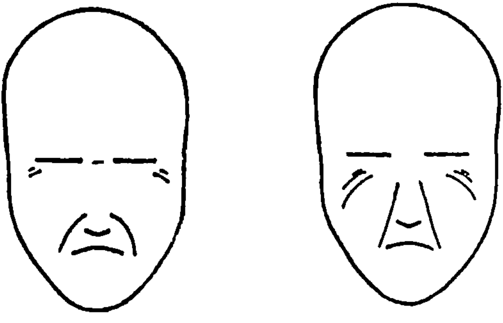
FIG. No. 25.

bone; from this origin it is directed obliquely downwards and inwards to be inserted into the deep surface of the skin of the commissure of the lips.

The triangular muscle of the lips (muscle of contempt).—This muscle depresses the labial commissure and, therefore, renders the line of the lips obliquely downwards and outwards. Again, it draws downwards the inferior extremity of the nasolabial furrow so as to render this furrow almost straight.

The expression produced by this change, if it is slightly marked, is that of sadness; if it is very much so, that of contempt. The partial closing of the eyelids usually helps to complete the expression of contempt.

The square muscle of the lower lip (muscle of disgust).—This muscle, partly hidden by the base of the preceding, arises like it, from the anterior part of the horizontal branch of the



A. B.

Fig No. 26.

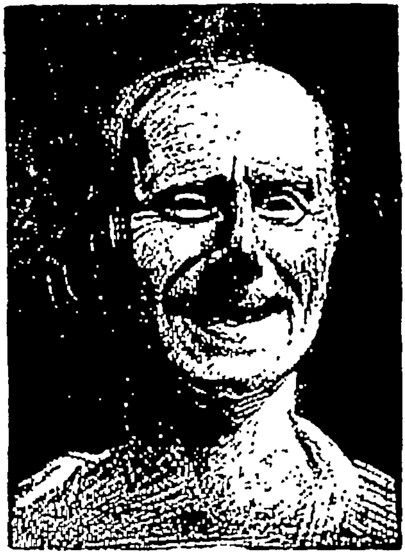
Fig A. The external common elevator of the lip is another name for the Zygomaticus Minor muscle (muscle of grief).

Fig. B. The internal common elevator of the upper lip is another name for the Elevator of the Upper Lip This is the muscle of grief and tears. It raises the upper lip but not the angle of the lip. It raises the center of the naso-labial fold. Its action is the reverse of the Zygomaticus Major, the muscle of laughter.

(After Duchenne.)

inferior maxillary; from this origin the fibres ascend obliquely upwards and inwards to be inserted into the whole length of the lower lip.

Platysma Muscle of the Neck.—On each lateral half of the anterior surface of the skin is extended a thin muscular sheet lining the skin. The platysma is attached below to the upper part of the chest, from which origin its fibres are directed obliquely upwards and forwards towards the lower jaw, to be inserted into the skin of the chin, of the lower lip, to the commissure of the lips and to the cheek. The more superior fibres are almost horizontal, extending from the skin of the region of the ear towards that of the labial commissure; to these superior fibres we sometimes give the name, but little justified, of the risorius of Santorini.



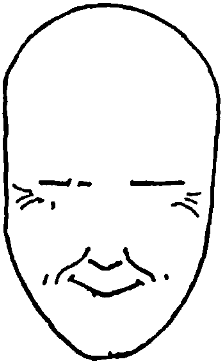
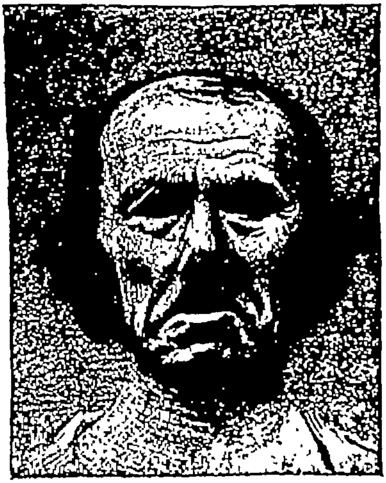


Diagram representation of *laughter.*

Great Zygomatic Muscle (expression of *gaiety* and *laughter*).

Fig. No. 27.



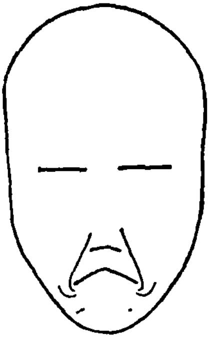


Diagram representation of the expression of *discontent*, of *contempt.*

Triangular Muscle of the Lips (expression of *discontent*, of *contempt*).

Fig. No. 28.

The platysma, which is not expressive by itself, adds its contraction to that of various muscles of the face, so as to give to the corresponding expression a character of terrible energy; the risorius of Santorini does not, therefore, produce the expression of laughter (of gaiety), but only that of grinning, of forced laughter, threatening, or sneering. The platysma, in all these cases, acts by depressing the lower jaw, slightly opening the mouth, and drawing the labial commissure downwards; at the same time, it marks a series of transverse folds on the skin of the neck. These elements are capable of giving to the face an expression of terror, which Leonardo da Vinci has well observed when, in his chapter on the mode of representing a person in a state of violent anger, he says: "Make the sides of the mouth in a bow, the neck thick and swollen, and marked with wrinkles in front."

We have just seen how the muscles of the face show the different emotions and, so, how an habitual facial expression can be an indication of a person's character. Modern investigators hold that the body type of a man also is an indication of his character. Some of their deductions seem at first a bit fantastic and make one think a little of the early writings of two hundred years ago.

Keith, Stockard, Davenport and Bolk are slowly bringing modern physiognomy toward the goal of the ancient writers, namely, the correlation between facial and bodily characteristics and character. What I have to say from now on is taken from Stockard. He *was thoroughly scientific as far as growth* is concerned. He did much work on the human fetus.

GROWTH OF THE BODY.

Two glands with their hormones, the hypophysis and the thyroid, control growth. The way they function determines whether we are skinny or fat. Jack Spratt and his wife owe their bodily differences to the different functioning of these two glands.

The normal amount of secretion from the hypophysis produces longer bones and a taller individual. When the secretion is sufficiently below normal, dwarfism is caused. Normal growth has to occur before the epiphyses close. Giving anterior pituitary extract has been known to cause five and a half inches of growth in twelve months.

VARIATIONS IN GROWTH.

The bull dog and a certain type of human dwarf with a broad face and a turned up nose owe their peculiar features to a derangement of the hypophysis. The base of the skull ceases to grow and becomes ossified. The upper jaw fails to grow. The head grows to the side, the nose fails to grow but the lower jaw, not being a part of the skull, grows out of proportion. The opposite condition and the stimulation of growth are also due to a disturbance of the pituitary. It is characterized by an excessive growth of bone in the linear direction. This is called acromegaly. The face is long. Giants are of this type. Among dogs, the St. Bernard and the mastiff show acromegaly with giantism. The blood hound shows acromegaly without giantism. His facial expression closely resembles the human acromegalic.

The opposite condition to giantism is responsible for true midgets. Midgets grow normally up to five or six years and then stop growing. They may or may not become sexually mature and often have infantile faces. Among dogs, the King Charles Spaniel, in shape, outline and expression is almost a picture of the human midget.

STOCKARD'S CLASSIFICATION.

Stockard recently classified the human face and the body into two types, the linear and the lateral. In the linear type, owing to a high rate of metabolism, — caused by a highly active thyroid gland, growth along the long axis of the body—from the tip of the nose down and back—greatly predominates over growth in the transverse plane. The linear type is the faster growing, the body is thin but not necessarily tall. The lateral type, owing to the low thyroid activity, is slow in maturing and the body is stocky and rounder in form; that is, the transverse growth components are relatively greater than in the linear type. Stockard's recognition of these two types was the result of long experimental work on the factors of growth during the embryonic development of animals.

The lateral type arrives at puberty a little late and is slow in differentiating. The larynx does not develop so suddenly, and is not so large. The voice is a tenor, not a bass. The fine tenor is a fat man (tenors, please stand), while the heavy bass is tall and thin (basses, please stand).

Stockard gives the character of each type, as follows; reading a person's character from his figure and face.

The lateral type is careful and painstaking and doesn't draw conclusions until a mass of detail has been accumulated. He is emotional and is easily moved to tears.

The linear type is tall and thin. He has difficulty in accumulating detail and in working a subject out thoroughly. He draws conclusions hurriedly. He is not emotional and is ashamed to cry. He has great self-control, and among savages the chief is always of this type. Among civilized people, the lateral type are often rulers of great ability. The lateral type are popular but do not see the great principles of the future. Presidents of the linear type are honored long after their terms of service, but are not popular during their terms of office. The lateral type of presidents are the idols of their time, but leave nothing to be remembered by.

CONCLUSION.

There is much justification, as I hope you have just seen, for the century-old belief that you can tell a man's character from his face. Intuition, however, or as Andy calls it, "a woman's ignition," is still a good guide in spite of all that science has learned and explained. Character shows so plainly in the face because the muscles of expression make the face wonderfully responsive to the emotions,—good and bad.

One more word, in closing. In case you did not notice it, I wish to remind you that in the face, the muscles of joy and sorrow lie side by side. In life, joy and sorrow are side by side. Nature knew what was coming to us, because she provided only two muscles for joy — but four for sorrow. If you are an optimist, put this in your pipe and smoke it.

127 Front Street.

THE USE OF CURARE[*] DRUGS IN ENDOSCOPY.[†]

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The introduction of curare-like drugs into clinical medicine as muscle relaxing agents has been of benefit to the patient, anesthesiologist and endoscopist. More information on indications and contraindications for their use as well as on techniques of administration is necessitated by an increased demand for these drugs for laryngoscopic, bronchoscopic and esophagoscopic procedures. The need for a proper, safe technique of administration has become apparent as more physicians use these drugs for such procedures. Many advantages and disadvantages present themselves in the use of curare. This paper is written in an effort to clarify the position of relaxing agents in the armamentarium of the endoscopist.

HISTORY.

"Curare" is the generic term applied to South American Indian arrow poisons. It was first brought to England in 1595 by Sir Walter Raleigh, and soon many stories were told of its paralyzing action and fatal consequences in animal and man. Then, except for the work of Charles Waterton (1812), who demonstrated that asphyxia is the cause of death by the drug, and Claude Bernard (1844), whose classical experiments showed the action of the drug to be on the neuromuscu-

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[*] The word "curare" will be used in this article in its generic sense to imply all of the natural and synthetic preparations having a pharmacologic action similar to d-tubocurarine chloride, which is the active alkaloid of crude curare.

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lar junction, curare fell into clinical obscurity. It became a toy for the physiologist and pharmacologist to demonstrate myoneural blocking action.

The successful introduction of curare into therapy began with Intocostrin, a preparation standardized by biological assay from an extract of chondrodendron tomentosum. Following the first reports of its effects in electric shock therapy[1] other indications for its use became recognized. Mixtures of curare alkaloids as present in the original preparation (Intocostrin) have been largely replaced by a single alkaloid, d-tubocurine chloride, isolated from the crude curare.[2] As a result, this drug is now widely used as an adjuvant in shock therapy, anesthesiology, endoscopy, tetanus, rigidity, spasticity and hypertonicity.

PHARMACOLOGY.

The classical work of Claude Bernard has established that curare acts on the myoneural junctions by preventing nerve impulses from reaching the effector muscle cells. The action is due to the elevation of the threshold to acetycholine, the chemical mediator which allows a nerve impulse to "jump" from nerve fibre to muscle tissue. Cholinesterase destroys acetylcholine; thus, physostigmine and neostigmine, being inhibitors of cholinesterase, permit acetylcholine to accumulate again to such an extent as to enable nerve impulses to overcome the curare block at the myoneural junction; hence, the cholinesterase inhibitors are antagonistic to curare, a fact which is made use of clinically whenever reversal of curare action is desired.

Therapeutic doses of curare have practically no effect on involuntary muscle and glandular secretions, but higher doses are believed to exert some blocking effect on autonomic ganglia.[3] The drug has no direct action on the central nervous system; consciousness, sensation, electroencephalogram all remain unimpaired, providing adequate respiratory exchange is maintained.[4] The administration of curare is followed by some fall in blood pressure which is at least partly due to the release of histamine, causing peripheral vasomotor dilatation. This histamine release is also the cause of undesirable and potentially dangerous manifestations, *e.g.*, bronchospasm and salivation; however, dilatation of the blood vessels as a direct consequence of decreased skeletal muscle tone also plays its part in causing fall in blood pressure. Sensory nerves are not affected by the drug.

Oral administration of curare has no effect since the drug does not penetrate intact epithelium; it is not destroyed by any of the digestive juices. After injection, the kidneys and liver are responsible for the elimination of the drug, but the exact mechanism of detoxification awaits further investigation.

Following the injection of curare, heaviness of the eyelids, diplopia and blurred vision are perceived. As curarization proceeds, the muscles of the face, tongue, pharynx, lower jaw, neck and back, and extremities become relaxed in about that order. With higher doses of curare the intercostals and finally the diaphragm are affected, resulting in respiratory arrest. The maximal effect is obtained about five minutes after injection. These signs recede in reverse order of their appearance, and within 30 minutes the maximal effect of the curare has disappeared.[5]

From this description it will be seen that the clinical picture is identical with the picture of myasthenia gravis, the manifestations of which will be greatly exacerbated by even minute doses of curare; the disease, therefore, is a contraindication to the use of any of the curare-like drugs.

Standardization of d-tubocurarine by chemical methods, in addition to biological assay which is based on the head-drop of rabbits, demonstrates that 3 mgm. (chemical assay) of the salt are equivelent to 20 units (bioassay).

CLINICAL USE.

The value of curare is its ability to produce controlled skeletal muscle relaxation. In patients who cannot relax when awake or who have muscular spasm, the use of curare greatly facilitates the process of intubation. With a relaxed patient, traumatic intubation, struggling movements, and expulsive coughing are usually minimized.

Laryngoscopy: Laryngoscopic procedures are greatly facilitated by the relaxation afforded by curare. In patients who have rigid neck muscles and in short, thick-necked patients, exposure of the anterior commissure is most difficult without relaxation. In laryngoscopy it is undesirable to introduce an endotracheal tube for artificial respiration when necessary, since the tube would be in the operative field. While it is true that the actual surgical part of the procedure can be discontinued if it becomes necessary to apply artificial respiration, nevertheless, such interruption often causes trauma to the operative site on the vocal cord; therefore, while relaxation is desired, deep relaxation to the point at which artificial respiration will become necessary must be avoided. As always, safety of the patient is the primary consideration. A satisfactory airway must be maintained at all times, and enough respiratory activity should be present to insure an adequate oxygenation.

One disadvantage in the use of curare in laryngoscopic procedures is that the patient may be unable to phonate adequately during operation on the vocal cords at a time when one would wish to see the results of the surgical procedure upon the cord. For example, in the removal of a polyp, it is desirable to view the tense, phonating cord on completion of the operation to determine whether the edge of the cord is entirely straight. Adequate local anesthesia and a correct dose of curare will permit this in the most satisfactory manner possible; however, larger doses of curare, or curare and pentothal, will make this impossible, with the result that the entire procedure has to be done on a loose, flaccid cord without determining the end-result as aided by phonation.

Bronchoscopy: Bronchoscopic procedures can, for the most part, be done under local anesthesia in adults, and without anesthesia in infants and children. Again, however, the anatomical problems of the rigid, short-necked patient or the patient with many protruding teeth and a high- narrow palate which make certain laryngoscopic procedures difficult, present the same problem in bronchoscopy. Thus, the relaxation afforded by curare simplifies the introduction of the bronchoscope in the difficult case. Adequate local anesthesia of the pharynx, larynx, trachea and bronchi is again an essential feature in patients who are to have curare. The substitution of pentothal for adequate local anesthesia is dangerous because of the violent expiratory spasm, laryngospasm and other reflex action which follow stimulation of the mucosa of the airway if the mucosa is not made insensitive by the local anesthetic agent.

Esophagoscopy: Esophagoscopic procedures present a number of varied indications for the use of curare. The anatomical difficulties of introduction of the esophagoscope are the same as those for laryngoscopy and bronchoscopy. The fullness of all pharyngeal structures in the obese individual is often an added problem. In esophagoscopy, rigidity of the neck not only makes introduction of the tube difficult but often makes it impossible to advance the tube into the stomach. This is due to the fact that the distal third of the esophagus points anteriorly and to the left, necessitating hyperextension of the head as the scope approaches the cardia.

In addition to the factors of instrumentation, curare greatly facilitates the removal of foreign bodies impacted in the esophagus. This is particularly true of such objects as bones and dental bridges which, because of their many sharp points, require maximal relaxation for safe removal during instrumentation. It is imperative, however, that an airway be maintained. Endotracheal technique is preferred in many cases for protection against possible laryngeal spasm and tracheal compression by the esophagoscope and the possibility of aspiration is decreased.

DISADVANTAGES.

The chief disadvantage of curare lies in its depression of respiration in overdosage. When the activity of the muscles of respiration is decreased, the diminished tidal volume and respiratory depression can place the patient in a dangerous condition, in that hypercarbia and hypoxia result. This state may cause pathologic alterations if allowed to persist. When the respiratory muscles are completely curarized, respiration ceases and, unless prompt combative measures are instituted, temporary or permanent damage or even death may ensue.

Curare does not produce sleep, sedation, analgesia, hypnosis, or amnesia. If these effects are required, other drugs must be used to attain these ends. There is no absolute protection against laryngospasm. Many clinicians are under the impression that a curarized patient is protected against laryngospasm; this is an erroneous and dangerous belief as this unfavorable reaction can and does occur.

Because of peripheral pooling due to decreased muscle tone, return circulation to the heart is diminished; this in turn produces decreased cardiac output with its resultant hypotension. Another disadvantage of some curare drugs is in their histamine-liberating actions, the most serious of which results in increased secretions into the respiratory tract, in bronchospasm and in hypotension.

COMBINED TECHNIQUES.

Other drugs used in conjunction with curare are the topical anesthetic agents and sodium pentothal, or similar sleep-producing agents. A thorough topical anesthetic with such agents as pontocaine or cocaine is mandatory.

The administration of pentothal with curare adds a very definite hazard of which both the endoscopist and anesthesiologist must be aware. There is an increased danger of laryngospasm with pentothal, especially in the lighter stages of anesthesia. Pentothal has a depressant effect upon respiration and circulation. An insufficient tidal volume is produced when respiratory depression is superimposed upon the already existant depression caused by curare; hence, the combination of pentothal and curare is pharmacologically bad and dangerous to the patient. Even in skilled hands this technique is most hazardous.

PREPARATION OF THE PATIENT.

A good history and a routine physical examination of the patient are assumed. In addition, the patient should be critically questioned concerning any previous endoscopic operative or anesthetic procedure, and an evaluation should be made of any untoward reactions The mouth should be examined for loose teeth or foreign bodies, and the mobility of the neck and temporomandibular joint should be established. The possibility of myasthenia gravis should be eliminated because in that disease curare is contraindicated.

As in any procedure that may produce respiratory arrest and circulatory depression, the following precautions must be taken: Equipment necessary to establish artificial respiration with oxygen should be on hand, and tracheotomy equipment and endotracheal tubes must be available for immediate use; intravenous fluids and vasopressor drugs may be necessary; Prostigmine, the pharmacologic antagonist for curare, is useful on some occasions and should be readily available; when Prostigmine is used, atropine is given to antagonize the secretory and other effects of the Prostigmine; in addition to all these resources, an anesthesiologist who is trained in the indications for and skilled in the management of resuscitation should be present.

The psychic preparation of the patient is a very important part of the technique. A short explanation of the procedure is of value. In bronchoscopic procedures he must be told that it will be impossible for him to talk because of the position of the bronchoscope between the vocal cords. When curare is used the patient should be forewarned of the relaxing effect of the drug, the sensation of weakness, heaviness, and heavy eyelids, inability to raise his head, and depression of respiration. Reassurance is equally important during the procedure itself and may be more valuable than administering drugs. It is mandatory to know the patient and obtain his confidence.

The preparation of a patient for an endoscopic procedure should be as thorough as for any surgical procedure. No food or liquids should be taken orally for a minimum of four hours before the scheduled time. Premedication is suited to the individual patient. Sedation in the form of a short-acting barbiturate such as nembutal or seconal is helpful. A dose of 1.5 to 3 gr. may be administered parenterally not less than two hours before the topical anesthetic. The barbiturate will also help to antidote toxic effects of the topical anesthetic agents.

Adequate doses of atropine or scopolamine should be prescribed. An average adult can tolerate as much as 1/100 gr. with no difficulty. This is necessary to decrease secretions and vagal tone.

Curare has a histamine-liberating factor and since histamine may have deleterious effects, *e.g.*, bronchospasm and hypotension. The liberation of this substance has only unfavorable features. If antihistaminic drugs are administered preoperatively, these detrimental factors will be held to a minimum. Antihistaminic drugs are useful only if given prophylactically and are noneffective if given after the curare. Benadryl, histadyl, pyrabenzamine or similar agents are probably satisfactory.

The use of morphine sulphate in premedication for these cases should be avoided. The common circulatory side reaction of morphine that may interfere with the procedure is diminished vasomotor tone with subsequent decrease in blood pressure. The respiration may be depressed. Nausea and vomiting occur frequently. Morphine is a parasympathomimetic drug and as such tends to enhance vagal reflexes and bronchospasm; therefore, if sedation only and not analgesia is indicated, it is wiser to use a barbiturate to obtain this end than an opiate.

TECHNIQUE.

A thorough topical anesthetic to include the pharynx, larynx, and trachea and bronchi is the most essential requirement. A simple technique is employed, using a minimal amount of anesthetic agent to accomplish a maximal effect. The pharynx is first sprayed with solution by means of an atomizer, using a topical anesthetic agent. Care is taken to direct the spray onto the posterior pharyngeal wall and base of the tongue. After three minutes the larynx is sprayed with the same agent, directing the atomizer tip toward the larynx as the tongue is held forward with gauze. After a second three-minute interval the larynx is visualized with the laryngeal mirror and 2 cc. of 10 per cent cocaine instilled onto the larynx and into the trachea drop by drop. If the patient is to have a bronchoscopic examination, the last cubic centimeter can be instilled as the patient leans first to one side and then to the other to allow the anesthetic agent to flow into each main bronchus. Unnecessary delay in starting the endosiopic procedure after this routine will result in a loss of operating time.

An intravenous infusion is started to provide a readily available channel for drugs or antidotes. The d-tubocurarine is administered, using the clock as a guide. Give an initial dose of 1 cc. (3 mg.) and wait three minutes; then give two-thirds of the estimated dose (0.13 mg. per kilogram of body weight) *and wait four minutes. Succeeding doses of 1 mgm.* should be given with caution and with three-minute periods of waiting. Enough curare should be given to depress the hand grip to the point where the patient can just squeeze one's fingers and is unable to raise his head; at this point administration must be stopped. Diaphragmatic movement still persists, and respiration is usually adequate. The respiration, eyelid tone and head-raising ability should all be carefully observed. Only the minimal amount of curare should be used to produce the required effect. Oxygen can be insufflated during the induction period.

As the drug is administered intravenously and has a long effect, it is much better to approach the required dose *slowly,* than to obtain an overdose with its subsequent hazards. The anesthesiologists should be allowed enough time to relax the patient gradually and safely. One should count on taking 15 minutes by the clock to get the patient ready.

During the procedure adequate respiratory exchange should be insured. Oxygen can do no harm in the preparation for and during endoscopy. As cyanosis might occur, this sign must be watched for, but its absence means nothing. A finger must constantly be on the patient's pulse to know what the circulation is doing and the other hand on the costal margin to determine respiration. This is especially important in a darkened room, in which case the anesthesiologist should also listen to the respiratory exchange.

The original dose will last at least 20 minutes. If more is needed at that time successive small doses can be given in order to maintain adequate relaxation. At the end of the procedure it is good protection against respiratory obstruction to place an airway in the patient's pharynx. Any secretions should be aspirated at that time.

The anticurare drug prostigmine must not be relied upon as an antidote to curare. Assured, adequate respiration is the antidote to curare. Drugs such as prostigmine and RO 2-3198* have proved very valuable. It is our choice to use a mixture of 1 cc. of the 1:2 000 prostigmine with 10 mgm. of RO 2-3198. This mixture seems to give rather prompt and lasting effect in antidoting curare.

DI-METHYL TUBOCURARINE IODIDE.

D-tubocurarine is the standard drug referred to in this paper; however a newer drug, Di-methyl tubocurarine [Metabine (R), Mecostrin (R)], has lately been used by the authors. Di-methyl tubocurarine, as with d-tubocurarine, has the advantage of having a pharmacologic antidote and can be repeated safely.

Di-methyl tubocurarine is superior in its safety as indicated by the therapeutic index. This is demonstrated by the fact that there is a substantial margin between the effective relaxing dose and a dose producing profound respiratory depression. The action of this drug can be more accurately predicted. Also, Di-methyl tubocurarine is better in that it has a shorter duration of action and has less histamine-releasing effect.

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*Anticurare drug in experimental state and not yet released. Product of Hoffmann La-Roche, Inc.

The ratio of dosage is 1 mgm. of Di-methyl tubocurarine to 3 mgm. of d-tubocurarine; therefore, in the curare induction for endoscopy a test dose of 1 mgm. is given, followed by a three-minute wait, then two-thirds of the estimated dose as calculated, followed by another period of waiting. At present, Di-methyl tubocurarine is considered the best of the group of relaxing agents and is the drug of choice.

OTHER RELAXING AGENTS.

Decamethonium Bromide (C 10) Syncurine (R): This drug has the shortest action of the group. It should be diluted before administration to obtain a more carefully regulated dose. Decamethonium bromide has the advantage of little histamine-producing effect. The disadvantages are many, however, and include no available antidote except artificial respiration and oxygen, noticeable blood pressure fall, and small therapeutic margin. Its action is the least predictable in controlled experiment.

Flaxidil: This drug is very similar to d-tubocurarine in duration of action, predictability and pharmacologic antidote. There are advantages not shown by the other drugs in the group. The blood pressure tends to rise instead of fall. Flaxidil seems to have a predisposition for the head and neck muscles, and this facilitates intubation without diaphragmatic depression. Anothed advantage is the depressant effect on the parasympathetic division of the autonomic nervous system.

SUMMARY.

The basic purpose of curare and curare-like drugs is to produce muscular relaxation and facilitate endoscopy. The use of these drugs is discussed. The necessary cautions in the preparation of the patient and the precautions to take in case of emergency are enumerated. A technique of administration is suggested. The pharmacologic properties of d-tubocurarine, Di-methyl tubocurarine iodide, decamethonium bromide and flaxidil are compared.

Clinically and experimentally, Di-methyl tubocurarine iodide is the drug of choice.

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REPAIR OF THE TRAUMATIZED LARYNX.*

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Prior to World War II there was a dearth of literature concerning injuries of the larynx. Textbooks universally either failed to mention this type of injury or hastily passed over it. The reason for this is the high death rate from these injuries in World War I and the sporadic cases of laryngeal injuries encountered during civilian practice. Between World War I and World War II there was still little to be found concerning these injuries in the medical literature; however, World War II served as a stimulus so that many more articles have appeared during the past six years than in the preceding 20 years. These reports[1-19] have contributed valuable information concerning laryngeal injuries, and experience in the treatment of such wounds is gradually accumulating. With the increased speed of travel by air as well as by land, and *with increased mechanical and chemical power, laryngeal* injuries which occur now, if they are not fatal, are usually far more extensive than previously.

Laryngeal injuries may be classified as lacerating wounds, incised wounds, penetrating wounds, contusions and crushing wounds. Such injuries have occurred as the result of striking the windshield, dashboard, steering wheel or rear seat in automobile accidents. Railway accidents usually produce crushing wounds; airplane accidents, crushing wounds, lacerations or burns. Injuries occurring in the household, on the farm, or during athletic events, altercations and suicidal attempts have also involved the larynx. Medically laryngeal injuries have resulted from high tracheotomy, endotracheal anesthesia or

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explosive anesthetics, delivery, irradiation burns or accidental severance of the recurrent laryngeal nerve. Thus, such accidents are an ever present possibility; therefore, the care of these victims is of interest not only to the laryngologist but also to the general surgeon whose responsibility is the first aid care and often the performance of lifesaving procedures in such cases. These patients usually die from asphyxia or hemic drowning. Asphyxia results from displacement of tissue, emphysema and compression, submucosal hemorrhage or hematoma blocking off the airway; drowning is due to injury to large vessels. The employment of emergency measures to prevent death from such complications usually falls to the general surgeon or resident on emergency call who, in an attempt to save the patient's life, may do a great deal of damage so far as later care is concerned.

Control of hemorrhage is as important as establishment of an adequate airway, because if either one is neglected for long, the outcome may be fatal. Hemorrhage may be controlled by clamping or pressure as first aid measures, and ligatures should be applied, if possible, but their application may be delayed for a more opportune time; however, the airway requires immediate attention. In such cases tracheotomy is truly a lifesaving procedure and often a race with time. The patient's head should be extended as far back as possible, after which the thyroid notch, prominentia laryngea and cricoid cartilage should be palpated. In dire emergency by an untrained technician the space between the thyroid cartilage and the cricoid cartilage is the easiest and most accessible point of entrance. A transverse rather than vertical incision should be made not only because in this way injury to either the thyroid cartilage or the cricoid cartilage may be avoided but also because there is less likelihood of damaging the inferior laryngeal artery, as the incision is made parallel to this vessel. High tracheotomy should always be a temporary procedure and should be corrected as soon as time permits into a low tracheotomy. The latter is the procedure of choice whenever possible and should be made low, as far from the site of injury as possible. This places the laryngeal cartilages at rest and facilitates later surgical repair if necessary by placing the tracheotomy wound as far away from the operating wound as possible; therefore, the incision should be made between the third and fourth tracheal rings or even lower in patients with long necks The thyroid isthmus should be split, bleeding controlled by continuous sutures and the pretracheal fascia sutured over the cut surface of the gland to the strap muscles and the muscles in turn sutured to the subcutaneous fascia This helps prevent emphysema and in the presence of a possible infection secondary to the wound helps to seal off the fascial planes adjacent to the trachea

Shock is the next consideration. Despite statements to the contrary, there is a greater degree of shock in these patients than would be expected from the extent of the laryngeal injury. Trauma to the larynx itself seems to produce this shock. Convulsions and early stages of shock are frequently seen in these patients, especially if attempts at surgical intervention are begun before the patient has been deeply anesthetized or if insufficient time is allowed between local infiltration and the onset of operation Then, too, these patients are extremely apprehensive, either because they *know they are expectorating blood or they are having difficulty* in breathing. Shock should be treated by establishment of an adequate airway, control of hemorrhage, reassurance, application of heat and administration of transfusions and warm infusions Morphine or other opiates are contraindicated except in small doses because of the possibility of decreasing respirations and depressing the cough reflex Sedatives in small doses given frequently are more advantageous at this stage and should be discontinued as soon as the patient begins to react from shock.

Following these emergency measures the larynx may be carefully examined In open wounds or in those partially healed the diagnosis of laryngeal injury can be made on external inspection In healed wounds there may be a tracheotomy because of narrowing of the lumen In recent wounds careful inspection should be made as much to determine the amount of tissue and cartilage remaining as a basis from which to begin reconstruction as to determine the extent of injury and loss of structure.

With the present use of the chemotherapeutic and antibiotic agents, the immediate care of these patients has changed. All structures should be preserved as much as possible. Debridement is indicated only in extremely contaminated wounds or in cases in which foreign material has become embedded in the tissues *en masse*. Even in these cases debridement should perhaps be incomplete, in the strict surgical sense, in order to preserve tissue. All structures that appear to have a chance of survival should be preserved and especially is this true of cartilage, which is extremely difficult to replace. If infection does occur, it is usually localized; in such cases soft tissues should be drained and cartilage removed as sequestra. It is surprising how often little or no infection will develop despite contamination, and when it does, how little devitalized tissue results. This is largely due to the use of chemotherapeutic and antibiotic agents.

Palpation will determine the amount of emphysema and its extent if present. With secondary swelling, the normal contour of landmarks of the neck may become obliterated. The cricoid, the cricothyroid space, the contour or the outline of the thyroid cartilage and thyroid notch as well as the thyrohyoid space may be palpated. The examiner should determine extent of laceration, displacement of cartilage, destruction of soft tissue and presence of foreign bodies.

Following inspection, indirect examination of the larynx should be attempted; however, even with cocainization, in many cases this procedure cannot be accomplished because of secondary swelling or distortion of the parts from injury. Nevertheless, when it can be accomplished, indirect examination often gives valuable information as to function of the remaining or injured structures before direct examination is begun; also, before direct examination is done both anterior and lateral Roentgenograms should be made. These will help to determine how much cartilage remains, whether the cartilages or hyoid bone have been fractured and in many instances the presence of opaque foreign bodies.

The next step in management of these patients is direct examination This is the most important phase of the examination As a rule it will give considerable information on the degree of injury and will help in formulating steps for repair or other treatment Because of the importance of this examination, suspension laryngoscopy is recommended (see Fig 1). By this procedure a complete picture of all the structures can be obtained through a wide open mouth with no part of the instrument in the field of examination, and most important of all, both of the examiner's hands are free to make a careful examination Furthermore, the examiner may sit before the patient and take time to make a thorough examination In this way the mouth, base of the tongue, pharynx and hypopharynx as well as the opening of the esophagus may be thoroughly visualized Next in order, the arytenoids,

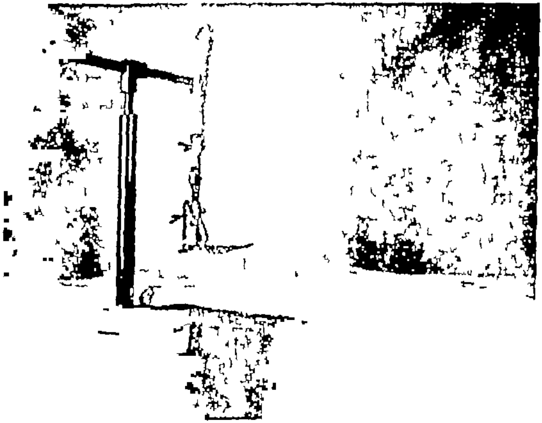

Fig 1A. The suspension apparatus consists of a suspension crane with a horizontal bar By means of two cranks the bar may be elevated or lowered transposed forward or backward. The suspension hook consists of a tongue spatula elevated at its tip for engaging the epiglottis, a flexible mouth gag and an arm with a hook for attachment to the crossbar Midway in this arm is a bat ring screw for changing the fulcrum and elevating the epiglottis.

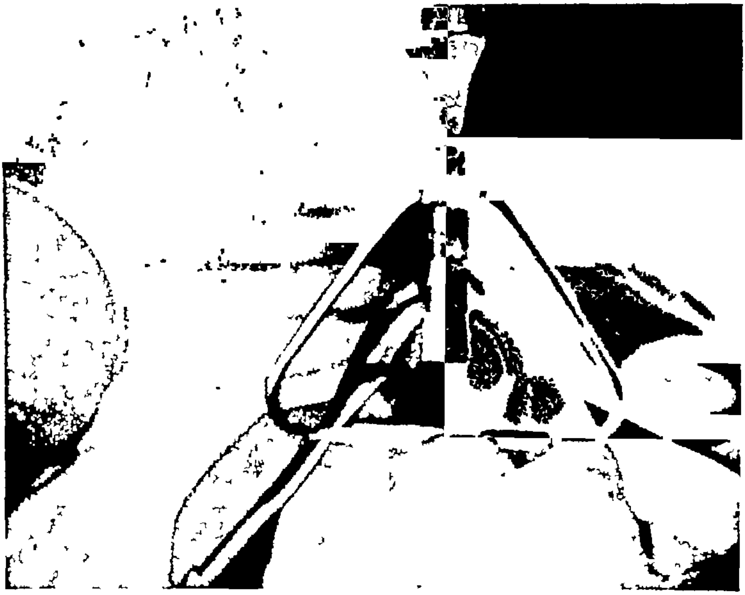

Fig. 1B The suspension apparatus in place showing the wide open mouth, the hypopharynx and opening of the esophagus as well as the interior of the larynx and vocal cords The importance is the ability to sit before the patient and have both hands free to make a careful examination.

ventricular bands, ventricles and vocal cords should be examined. The cords may be retracted and the area beyond studied more conveniently and as completely as with any other method. Fixation or displacement of the arytenoids, paralysis of one or both cords, cicatricial bands, webs and the degree of stenosis can be noted.

By means of external examination and palpation, Roentgenography, indirect examination and finally direct visualization, formulative plans can be made for reconstruction. Although no two cases are alike and each must be managed individually, there are certain procedures which apply to all cases. After an adequate airway has been established, and hemorrhage and shock have been controlled, it is necessary to determine whether the esophagus has been injured. If this has not been determined prior to examination, it must be established at examination. By means of suspension it is easy to introduce the esophagoscope to the level of the suprasternal notch; in other words, the area posterior to the larynx and trachea should be visualized at the time of direct examination of the larynx.

Esophageal injuries should receive attention first. Fortunately, the esophagus is only rarely injured and usually this occurs only in the severest types of cervical injuries. If the injury is small and a fistula is present, this should be excised and the wound closed, but a feeding tube should be left in place from five to seven days. If the esophagus has been severed completely, it should be closed from the posterior surface to the anterior surface; the musculature of the posterior surface should be sutured first, then the mucosa of the posterior and lateral walls, followed by the mucosa of the anterior wall, and lastly, the musculature of the anterior wall. The feeding tube should be left in place for 10 days and the wound carefully examined for stricture before the laryngeal injuries are treated if these happen to be extensive. These wounds are usually stab wounds made with a blunt or sharp instrument and as a rule there is little loss of laryngeal or tracheal tissue so that they may be repaired at the same time; however, if a portion of the esophagus has been destroyed, its repair should

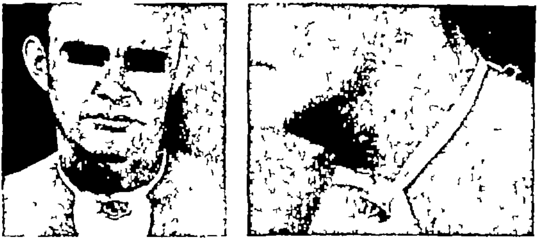

Fig 1. Contusion of the thyroid cartilage with simple linear fracture of the alar, complicated by hematoma involving the false cord with obstruction to the airway Tracheotomy was necessary The hematoma was large, the surface was scarified and a deep puncture made. Drainage and absorption were slow but the swelling subsided. Decanalization was accomplished, the voice remains good, the airway normal.

be completed before reconstruction of the larynx is begun. The esophagus should be repaired by means of a skin flap brought over from the surface of the neck or a tube graft from the shoulder or back, as indicated. If a flap is used, a two-stage procedure should be employed. The flap should be sutured first in the bed of the esophagus and attached above and below to the severed ends of the esophagus. This should be left alone for two months after it has taken. Then the flap should be cut, folded over a large rubber tube (size 48 to 50 French) and sutured so that the lumen is composed of epithelium and the raw surface is external. This may be allowed to heal by itself or healing may be hastened by means of skin grafts. It may be necessary to dilate this tube, especially at

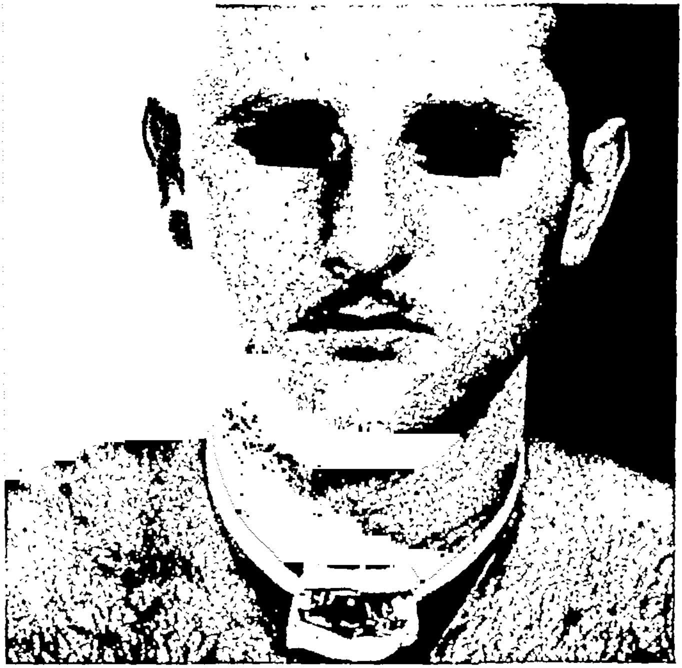

Fig. 3. Stab wound of the neck, piercing the thyroid cartilage with laceration of the mucosa of the larynx. Hemorrhage and secondary edema necessitated tracheotomy. The laceration of the larynx was sutured intrinsically by means of suspension laryngoscopy and the external wound was closed with insertion of a silkworm drain. Healing was uncomplicated. The tracheotomy closed, the patient has a good voice and the airway is adequate. The cord was not injured.

its attachment above and below to the esophagus, but surprisingly enough this is uncommon if the lumen is made large enough from the start

After closure of the esophageal fistula or repair of the defect, attention may then be centered on the larynx Low tracheotomy should be performed first if this has not already been done. Then repair of the larynx may be begun. A frequently overlooked injury is fracture of the hyoid bone This can be determined by the expectoration of blood, injury of the pharyngeal mucosa, disturbance of phonation, pain when the

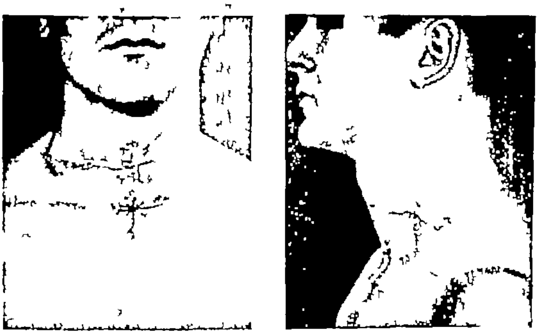

Fig 4 Gunshot wound of the neck involving the esophagus posterior portion of the wing of the thyroid and the arytenoid. Following tracheotomy the injury to the esophagus was repaired. After this healed and it was determined that the lumen was adequate the larynx was repaired Laryngofissure was performed, the scar of the arytenoid and false cord on that side was removed and skin was grafted As soon as the skin graft had taken cartilage was grafted to support that side of the larynx The patient now has a good airway and a surprisingly good voice considering the extent of injury and loss of tissue.

tongue is extended or moved from side to side, and immobility of the epiglottis Roentgenograms will confirm the diagnosis. If the fractured hyoid bone is associated with much displacement of fragments and it is impossible to reduce the fracture, it may be wired; however, in the majority of cases, by the pull of the muscles the fractured segments will reduce themselves. The simple, single fracture should be left alone, as healing will occur and symptoms will be relieved in a relatively short time.

Laryngeal injuries have been divided into those above the hyoid bone, those of the thyroid cartilage, those through or below the cricoid cartilage and those of the trachea; however, because of the relative shortness of the neck, many of these injuries overlap or may even involve all structures. As a

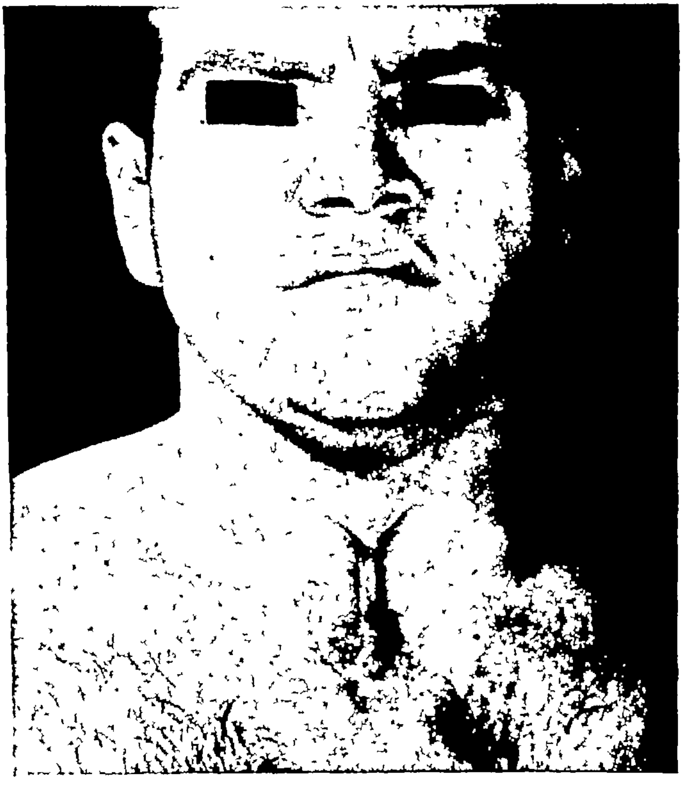

Fig 5 Crushing wound of the neck involving the thyroid cartilage Following tracheotomy, laryngofissure was performed; the segments of the thyroid cartilage were restored to position and held in place by means of a laryngeal stint. Secondary infection resulted in loss of only a small segment of cartilage. This infection was drained as soon as detected and the chemotherapeutic dosage increased. The patient has good support and a good airway, but only fair phonation due to laceration of the mucosa at the time of the accident. The stint resulted in scar fixation of the laceration intrinsically and the cords are only moderately involved.

working classification, these injuries may be divided into: *1.* wounds from without, involving the cartilages or hyoid bone without intrinsic laceration or injury, *2.* wounds producing laceration or injury to the interior surface of the larynx, *3.* wounds producing little or no external injury but causing displacement and laceration of the interior surface, and *4.* a combination of the first two or all three of these types of injuries.

Wounds of the frontal surface of the neck with no damage to the cartilage and without intrinsic laceration should be debrided and sutured. During debridement as much tissue as possible should be conserved because the structures encountered in this location are so compact and so closely related that extensive removal of tissue often does more harm than good. Following debridement, sulfonamides should be implanted in the wound and the wound sutured. The systemic administration of penicillin is also indicated.

Wounds of the frontal surface of the neck heal without undue complications and the prognosis is good; however, extensive debridement may convert a simple injury into a complicated one or may result in paralysis of the cricothyroid muscle through injury to the external branch of the superior laryngeal nerve.

Next in severity are wounds of the thyroid cartilage, which may be either open or subcutaneous. If there is no intrinsic injury, the prognosis as to voice and eventual adequate airway is good, since laryngeal edema usually develops immediately after injury. Recovery is rapid; however, these patients should be examined frequently and watched closely for the possible appearance of edema or hematoma, either of which may interfere with breathing. At the first signs of respiratory distress tracheotomy should be performed. This prevents tugging and possible displacement of the fractured thyroid cartilage and places the larynx at rest; the edema then usually rapidly subsides and the hematoma quickly disappears. These patients should also be carefully observed for the development of perichondritis or chondritis, which is not easily detected, since often the only positive sign is persistent, slight tenderness, later becoming progressively more severe. Usually this is localized and the temperature, blood picture and sedimentation rate remain normal. There is still considerable controversy regarding the time and advisability of operation in the presence of this complication. It has been my experience that more of the cartilaginous structures can be preserved by early operation with drainage and chemotherapy than by waiting for complete walling off of the infection and slough.

Injury to part of, or shattering of the cricoid cartilage presents a serious problem. There is likely to be some form of web present and often complete cicatricial stenosis of the larynx. At the point where there is loss of support the larynx collapses and this may be extensive. As a result of shattering the stenosis is much more severe than the stricture resulting from high tracheotomy and repair is more extensive and prolonged. In such cases cartilage graft, or acrylic or tantalum implant, preceded by skin graft, is necessary.

Injuries involving the integrity of the laryngeal cartilages are, next to complete excision of the larynx, the severest types of laryngeal trauma and are often fatal. In such cases establishment of an airway by skin grafts, graduated molds and cartilage grafts taxes the ingenuity of the surgeon and the patience of the injured. If the epiglottis and arytenoids are not injured, an airway may be established, but if these structures have been severed or destroyed, a functioning larynx cannot be obtained. Without the epiglottis and with paralysis of the arytenoids an airway may be established, but liquids will be repeatedly aspirated. Correction of this causes the airway to be inadequate; however, these patients do well with a tracheotomy tube, are able to speak, and with the narrowed airway above the tracheotomy tube, are able to eat and drink with no discomfort.

The most extensive laryngeal injury is one which also involves the trachea. Frequently, in such cases, in addition to the esophagus, the cervical vertebrae are injured. These patients usually die at the site of injury and rarely reach a hospital. If they do not die, they should receive the same treatment already described except that it should be more extensive. Repair of the trachea has been accomplished experimentally with fascia lata. Acrylic and tantalum molds have been used for support but these have not been accomplished on the human.

In those cases involving shattering of the cricoid cartilage, injury to the entire larynx and injuries of the larynx and trachea, the esophagus is usually also involved; the care of this structure has previously been discussed.

Injuries involving the intrinsic surface of the larynx vary from slight laceration to complete cicatricial stenosis. If the laceration is small and emphysema is not present, it is wise to leave it alone; however, if emphysema is present and progressive, tracheotomy should be followed by suturing the laceration either intralaryngeally with the use of the suspension apparatus or by means of laryngofissure after subsidence of the emphysema. Hematoma will absorb, but if it is extensive enough to block or embarrass the airway, following tracheotomy the membrane should be scarified and one or two deep punctures made to facilitate drainage; otherwise, complete absorption will not take place, in which case fibrosis and distortion of the interior of the larynx will result. This in turn causes restriction in the ability to speak and at times interference with respiration because of fixation of one or both vocal cords.

If a web is present, it should be incised anteriorly and a stint inserted. After the tissue around the stint has healed, the web may be excised; if it is not excised, it will have a tendency to recur. If there is a stricture with a lumen and the scar is not too thick or too extensive, it may yield to repeated dilatations. If the scar is dense and thick, however, core molds should be inserted gradually, the size being increased as the scar tissue retracts and yields to the pressure. These molds are well tolerated by the larynx and relatively comfortable to the patient after once being inserted.

Complete stricture, if thin and not too extensive, can be incised with the aid of the suspension laryngoscope and core molds inserted in the manner described in the preceding paragraph; however, if the stricture is dense and extensive, laryngofissure is the procedure of choice. The scar tissue should be completely excised and a skin graft inserted around a mold the size of the uninjured lumen. This should be left in place for two months and removed through the mouth. It is sometimes necessary to insert core molds following this, but often if left in place for this length of time, these are not necessary.

Complications following laryngeal injuries are prone to develop. The fascial spaces, opened from the time of injury, provide pathways for cervical and mediastinal infections. Emphysema may develop at the time of injury or later from an open type of tracheotomy in which the fascial planes are not sealed off, or from too tight suture of the external wound and internal lacerations. Pneumonia is an ever present danger because of the likelihood of aspiration. The mucous glands of the trachea tend to keep the skin graft moist, and the graft, being a foreign body, is liable to slough. Molds the size and conformity of the lumen help to prevent this complication but it is an ever present possibility during the early stages of skin grafting. The operative site within the larynx may become secondarily infected; in such cases the structures must be taken down, the infection overcome, and the procedure begun again. Perichondritis and chondritis, ever present hazards, require immediate drainage in order to preserve as much cartilage as possible. Prophylactic measures against all these complications include suction at the bedside, administration of chemotherapeutic and antibiotic agents both before and after operation, transfusions when indicated, general high protein and high caloric diet, and the use of a closed respiratory system during anesthetization and operation.

Of final importance is rehabilitation of the patient. If no attempt is made to improve phonation, a severe psychosis may develop. Breathing is not enough; these patients also want and should be able to talk. Following completion of surgical treatment, if necessary or advisable, the patient should be sent to a speech center or speech correction school before final discharge.

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PRINCIPLES OF THERAPY IN OTITIS EXTERNA.*

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World War II served to overturn two previous concepts concerning otitis externa. The first, which came as no surprise to otologists who practice in the South, was that it was a disease of low frequency and little importance. The second was that otitis externa is chiefly a disease of fungous origin. Actually, otomycosis proved to be a great deal less frequent than bacterial otitis, and fungi are now believed to be of only minor significance in the causation of the disease. The therapeutic implications of this etiologic reversal I shall return to shortly.

The chief reason for the confusion which presently exists concerning external otitis is the conscious or unconscious attempt to gather all its manifestations under the same pathologic umbrella. In reality, external otitis is a general name for a number of different diseases whose chief point of resemblance is that they are located in the external auditory canal. Naturally, since the various observers are often not talking about the same conditions, published reports of the results of similar modes of therapy often vary from miraculous successes to complete failures. The basic confusion, of course, stems from the fact that treatment has been instituted without proper etiologic studies, or without a sufficiently wide experience to justify the expedient of treatment without such an investigation.

It seems fair to add that additional confusion is caused by the rather general medical failing of rushing into print with

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reports of agents which often have not been tested for long enough periods of time to warrant any conclusions at all, let alone sweeping statements concerning their efficiency.

These pessimistic observations do not mean that the therapy of external otitis has not been greatly clarified within recent years; it has been. Among the extremely valuable contributions to this subject are the studies of McBurney and Searcy,[1] in 1936, the continuing contributions of W. D. and E. K. Gill,[2-7] and the recent investigations of Senturia and his associates.[8-13] No one should attempt to treat this disease without familiarizing himself thoroughly with these studies; moreover, the introduction of efficient chemotherapeutic and antibiotic agents and of the newer chemical and synthetic agents has eliminated the necessity for the use of many of the agents formerly employed, often empirically and often with no very valid indications for their use.

DIAGNOSTIC PROCEDURES.

The treatment of external otitis, regardless of the variety, will be greatly simplified if certain steps are followed routinely in its management and if certain general principles form the basis of every therapeutic regimen.

The first step in the management of any case of otitis externa is: *1.* a careful history, with special attention to possible etiologic factors, *2.* a general otolaryngologic examination, and *3.* a careful local examination, supplemented by magnification. The data thus secured usually permit an accurate clinical diagnosis, though it must be remembered that the clinical picture will vary according to the duration of the disease and its treatment before the patient is seen.

As an academic matter, laboratory studies are desirable in every case. In private practice, for a variety of reasons, including the cogent one of expense, there must be some compromise with the ideal. Fortunately, it is usually possible to determine by clinical observation, particularly if gross inspection is supplemented by magnification, what sort of infection one is dealing with, and it is entirely proper to begin treatment on the basis of the clinical impression, with a considerable saving of time as well as of expense. This short cut, however, is permissible only for otologists who have had a large experience in otitis externa. Competence in clinical diagnosis comes only with experience, and treatment without laboratory investigation is not recommended for those who do not possess it.

If the patient's failure to respond promptly to adequate therapy indicates that the clinical diagnosis was not correct, laboratory studies, including both smears and cultures, are instituted without delay. As Gill[6] has emphasized, some molds are slow-growing, particularly the types common in chronic or recurrent otitis externa, and cultures should not be discarded for a minimum of two weeks. Fortunately, the identification of the species of fungus is all that is necessary for therapy; only occasionally is further differentiation required. Two other precautions come to mind from my own experience: the hands should be washed thoroughly with one of the new antiseptic soaps before the culture is taken, and sterile cotton (either autoclaved or secured from the center of a commercial package) should be used for taking it.

GENERAL PRINCIPLES OF THERAPY.

With these fundamental considerations out of the way, we may now proceed to the principles upon which the treatment of otitis externa should be based.

1. The first principle of therapy is that whichever form is employed, it must be related to the etiology of the case under treatment. It is now generally recognized that the majority of cases are of bacterial origin. Not more than 15 or 20 per cent, at most, are of fungous origin. Aspergilli are apparently responsible for most cases in this group, though these fungi can often be obtained on routine cultures of normal ears and their mere presence does not imply that they are pathogenic. Staphylococci are responsible for a considerable number of bacterial infections, but there is a surprising uniformity in bacteriologic studies, both military and civilian, to the effect that one special organism, Pseudomonas, is the predominant organism, or the only organism, present in most cases. Pseudomonas is not present in normal ears, and it is significant that in many studies in which it was found in them, otitis externa later developed while the patients were under treatment for contralateral infections.

2. In addition to the application of therapy on the fundamental principle that an infection is of bacterial or fungous origin, other etiologic possibilities and predisposing causes must also be eliminated or counteracted. It is true that nothing can be done about climate and humidity, the two most important predisposing causes, but bad hygienic habits, lack of cleanliness, and failure to keep the ears dry can readily be corrected and will go far to overcome conditions which cannot be changed. The ears can be protected against trauma, chiefly trauma introduced by the patient himself. Other causes which must be investigated and corrected include anemia, vitamin deficiencies, and various allergies, of which more will be said later. A possible psychogenic origin is surprisingly frequent. In the occasional case of otitis externa one or another of these causes is solely responsible. In many instances they are present *in addition to bacterial or fungous invasion. In any event,* treatment will be less than adequate when they are not sought for and corrected.

3. Judgment should be used in the selection of therapeutic agents. As Senturia[14] has well expressed it, only those which are worthy of clinical trial should be selected. Those which have no relationship to the conditions present should be rejected. There should be, in short, bacterial and fungous cross-matching. If more than one organism is present, the predominant organisms should be treated. If these principles had been borne in mind, a large number of agents used in the past in the treatment of otitis externa would never have been tested at all, and many of those presently employed would at once be discarded. A review of the literature indicates how little attention has been paid — and is still being paid — to this fundamental precept.

In the same connection, it should be emphasized that whenever it can be avoided, therapeutic agents should be used independently and not in combination, if only for the reason that agents used in combination may cancel each other out.

4. Until quite recently, little attention has been paid either to the pH of the external auditory canal or to the pH of the solutions applied to it. The important studies of Fabricant and Perlstein[10] have shown that these are matters which must be taken into consideration in all therapy. It is not uncommon, in fact, to find that the difference in efficiency of two apparently similar agents can be explained by the fact that one produces or enhances a state of acidity, which is the normal state within the canal, while the other produces or enhances a state of alkalinity, which is unphysiologic and which provides optimum soil for the propagation of infection. Behind every case of external otitis, I believe, are two factors: interruption of the continuity of the epithelial lining and alteration of the pH from the acid to the alkaline side. Once these factors occur, bacteria and fungi have their great opportunity, and it is surprising how often their properties are activated by the use of solutions whose pH is on the alkaline side.

5. Chemotherapy and antibiotic therapy have, paradoxically, lost some of their usefulness because they have been so widely used. Sensitization is now a possibility to be borne in mind in all topical applications. Dermatologists contend that penicillin cannot be used locally much longer than five days without the appearance of erythema, vesiculation, and other evidences of sensitization. I think this is true of all other topical applications. This complication, in fact, has kept me from using, except in very occasional cases, some antibiotics of established usefulness, as well as many of the newer agents such as isopar. A limited experience with them has indicated that patients are highly sensitive to them and that their efficiency would be counterbalanced by this annoying and sometimes serious complication.

There are other reasons for employing antibiotic therapy cautiously. In the first place, the new chemotherapeutic and antibiotic agents are useful, and indeed invaluable, in conditions which are far more serious than otitis externa, or are actually lethal, and their careless use for what is, after all, a minor disease may destroy their usefulness for the special patient in some later emergency. In the second place, all strains of organisms do not respond to agents which are ordinarily effective against the species to which they belong. Some strains are originally resistant and some develop resistance in the course of treatment. When a lack of response to a properly selected agent is observed, cultures should be repeated and sensitivity tests run, to determine to what agent the organism is sensitive, and in what concentration. Drug-resistance to special strains, incidentally, probably explains some of the contradictory reports issued by observers of equal competence and reliability.

6. Otologists who are not familiar with the studies by Senturia and Doubly[11] on the importance of the vehicles used for the agents selected for the treatment of otitis externa would do well to familiarize themselves with them. They showed, for instance, that the incorporation of streptomycin, penicillin or mixtures of these two agents in water-insoluble vehicles results in the complete inhibition of their antibacterial effect against certain organisms. When they were incorporated in water-soluble vehicles, with the exception of propylene glycol, this loss of activity was not observed. Others have contributed along the same lines. Zimmerman,[14] for instance, pointed out that sulfa drugs are released only slowly and in minute quantities when they are incorporated in vaseline and lanolin bases.

One's own clinical experience is not necessarily in agreement with all these observations. Clinically, for instance, I have not found, as Senturia and Doubly's studies showed, that oily vehicles interfere with the fungicidal activity of cresatin and thymol, nor have I found, as Fowler[15] did, that better results were secured with sulfamylon when it was employed in 1 per cent solution of methyl cellulose rather than in carbowax or in aqueous solutions. Nonetheless, the studies to which I have briefly alluded suggest that when poor results are secured with agents which are effective *in vitro* against particular strains of organisms, it might be well to look into the vehicles in which they are being applied.

7. Whatever drug is selected should be used for as brief a period as possible. It requires nice judgment, of course, to decide what is long enough and what is too long, but it is a decision which nonetheless must be made. Long-drawn-out treatment is unnecessary in most cases and, as already pointed out, may be harmful. The normal physiology of the external auditory canal presupposes a dry, intact, horny layer of skin, and treatment should be designed, as far as possible, either to maintain it or not to destroy it any longer than is necessary. The skin is often damaged by the use of wet dressings and ointments, which should always be regarded as temporary measures and discontinued as soon as possible. Strong chemicals may cause a dermatitis to be superimposed on the original bacterial or fungous infection. Desquamation may be a necessary part of therapy, but when the need for it is past, measures should be taken to restore the normal status. In addition to the cessation of active treatment designed to eradicate the infection, the use of a mild coal tar ointment which also contains salicylic acid (such as pragmatar) is often necessary to bring the skin back to normal.

8. Whichever drug may be selected, in the light of the principles just discussed, must be applied in the light of certain other principles. It must be prepared in high enough concentration to affect the organisms against which it is used; there is no magic in solutions which do not meet this requirement. It must be applied in sufficient amounts to reach all parts of the external auditory canal. Contact between it and the diseased tissues must be close and intimate, which is not possible unless the canal has been thoroughly cleansed. It must, as just pointed out, be applied sufficiently long to effect the desired results but not long enough to induce sensitivity. It must be promptly discontinued and replaced by some other agent when it is evident that it is not accomplishing results, though the trial must be long enough for it to exert whichever therapeutic effect it is capable of.

9. Theoretically there are numerous sound arguments *against letting the patient with otitis externa participate in* his own treatment, and I grant Fowler's[15] contention that an analysis of cases may contain errors if the otologist has not been responsible for all the therapy. For practical reasons, however, it is simply not possible to see all patients with otitis daily, let alone several times daily, which is how most forms of therapy must be carried out.

This circumstance means that the physician must be extremely careful in demonstrating and explaining to the patient exactly how therapy is to be applied. He must also emphasize to him the importance of doing exactly what he is told — carrying out treatment at the precise times specified, occupying the position demonstrated, and keeping the solution in the ear for the time required. In my own office we avoid misunderstandings by giving the patient written instructions on all of these matters.

My own experience, which I have no doubt is duplicated by others, is that patients are much more willing to carry out treatment, and to carry it out more accurately, if the method is made as simple as possible, and also, if I may so express it, as attractive as possible. Dyes such as gentian violet and merthiolate are conspicuous. Ichthyol ointment and Castellani's paint are messy. From the otologist's standpoint they have the additional, sometimes serious, disadvantage of obscuring the progress of the disease. It is true that sometimes they are necessary. Gentian violet, for instance, together with potassium iodide by mouth, is a very effective treatment of Monilia infections. It is also true that in McBurney and Searcy's[1] studies some of these agents, such as Castellani's paint and merthiolate, rated rather high in the scale of efficiency in otomycotic infections. It would, therefore, be necessary to use them, in spite of their disadvantages, if nothing else were available; but equally efficient agents, which lack these objections, are now available, and there is seldom any necessity for using undesirable dyes and ointments.

10. Only in the exceptional case is hospitalization required for external otitis. As a general rule, the patient can be completely ambulatory or can rest at home between treatments. Occasionally a patient with severe furunculosis caused by a mixed infection, of with perichondritis, is hospitalized for parenteral antibiotic therapy. Misconceptions which have arisen on this point are probably attributable to the military experience, in which hospitalization for external otitis was fairly frequent. The explanation, of course, is that the circumstances of military and civilian life are widely different, and that certain regimens and precautions entirely compatible with the continuation of normal civilian occupations are often completely impractical for military personnel.

It is perfectly possible to carry out most local treatments and manipulations in the properly equipped office. Sterile precautions should be employed and manipulations should be limited to those which are absolutely required, and should be as gentle as possible, since the superimposition of additional trauma may greatly aggravate the condition and may introduce fresh infection.

ELEMENTS OF THERAPY.

To turn now to the active treatment of otitis externa, it is divided into several component parts, including *1.* the relief of pain or discomfort; *2.* the cleansing of the external auditory canal; *3.* the elimination of the causative agent and the elimination or control of the predisposing causes which permitted it to become established; and *4.* the restoration of the external auditory canal to a normal physiologic status.

My own preference, if it seems that the application of heat by means of a light will not be sufficient for the control of pain, is the use of small doses of X-ray. The exact mechanism by which results are achieved may still be uncertain but there is no doubt of the effectiveness of this method. Sometimes relief is obtained in as little as three or four hours, and well over half of all patients require only a single application.

I have used this method of relieving pain for a number of years. At first dosages varying from 42 to 62 r. were used. Then the dosage was gradually and cautiously increased, and at the present time I use 100 r. daily until relief is obtained, though in no case does the total dosage exceed 300 r. In addition to the relief thus secured, preliminary X-ray therapy seems to shorten the duration of treatment. The application, of course, must be made by an experienced radiologist, with the proper precautions, and Gill and Gill's[7] warning is timely, that repetition of the method in recurrent attacks is dangerous because of the ease with which the function of the ceruminous glands can be destroyed.

X-ray applications, in addition to relieving pain, are useful when cellulitis is present They may be supplemented by wicks saturated with some such agent as Burow's solution (1 per cent aluminum acetate).

I am one of those who believe that if it were practical to see patients daily, which of course it is not, it would be possible to clear up most cases of otitis externa merely by repeated daily cleansing. I am aware that some reports in the literature fail to bear out this belief, but, on the other hand, the cures obtained by Senturia and Broh-Kahn,[10] for instance, in control cases in which only carboprop was used do seem to indicate the possibilities of simple cleaning in simple cases. My own opinion, in fact, is that careful cleansing explains the good results reported with certain agents which simply could not in themselves have achieved the cures attributed to them.

Be this as it may, there is no question that almost without exception observers who report good results from any method of treatment emphasize the necessity of cleansing and drying the external auditory canal thoroughly before any applications at all are made to it. There are a number of good reasons for this phase of therapy: *1.* Moisture, maceration, and the accumulation of epithelial debris and dead tissue all offer else were available; but equally efficient agents, which lack these objections, are now available, and there is seldom any necessity for using undesirable dyes and ointments.

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Opinions differ over the best method of cleansing the canal. Some prefer irrigations, some prefer suction because they fear the introduction of fluids. I think that this fear, providing that the ear is thoroughly dried afterward, is unfounded.

My own plan, in the usual case, is to cleanse the ear gently by suction, first using the smallest size Frazier tip, and near the attic using a tip devised in my office for this purpose. This tip is malleable and, therefore, can be bent to reach inaccessible areas. Finally, the canal is irrigated with an ounce of 95 per cent alcohol and is thoroughly dried. For the irrigation I use the 2 oz. DeVilbiss bottle, which is easier to handle than a syringe and use compressed air for both irrigating and drying purposes.

This is not a procedure which can be hurried. It must be done deliberately, carefully and very gently. If it is done in this way it is not painful, though it may take as long as 15 or 20 minutes. Lighting must be adequate. If edema is so great that the initial manipulations are limited, a wick is inserted for 24 hours, saturated with Burow's solution, which I prefer because the pH (4.4) is on the acid side. At the end of 24 hours the desquamated membrane can usually be removed manually without difficulty.

Like most other otologists, I believe that cerumen should be removed from an infected ear, along with all other debris. Philpott and Chessen[18] comment rather bitterly on the difficulty of educating both soldiers and medical personnel to its "beneficently bactericidal" properties, but too much evidence exists that bacteria and fungi can grow on wax to make it wise to leave it in an infected ear.

A few words might be said about irrigating solutions. Some otologists feel that the solution remains in contact with the tissues too brief a time to make anything but water worthwhile. In that case, tap water is as good as anything, though it might be well to follow it with alcohol. Some prefer hypertonic salt solution. Others regard it as irritating and use isotonic solution. ST 37 and sodium bicarbonate have been recommended. Many prefer hydrogen peroxide. In McBurney and Searcy's[1] studies a 3 per cent aqueous solution of hydrogen peroxide occupied fifth place as a bactericide and twenty-sixth place as a fungicide. It is undoubtedly a valuable agent in both bacterial and fungous otitis externa, especially because of its ability to dissolve cerumen, crusts, scales and pus. It has no sensitizing properties, and when it is used in the proper concentration it does not harm the skin.

My own preference, as I have indicated, is for alcohol in 95 per cent concentration. It is interesting to note that alcohol has been used in otitis externa since 1874, and that Ballenger, in 1909, called it the remedy *par excellence* for fungous infections.[1] McBurney and Searcy[1] found that, regardless of the concentration employed, alcohol had no effect on any variety of Aspergillus on which it was tested. It is, nonetheless, an extremely useful cleansing agent and it has the great advantage that if any quantity should inadvertently be left in the ear, it promptly evaporates.

McBurney and Searcy,[1] in 1936, tested 69 drugs and combinations of drugs which had been recommended for use in otomycosis up to that time. Offhand, I would say that at least as many drugs and combinations have been recommended since that date.

Before going on to the treatment of special types of otitis externa, we should be reminded that the classification of the disease is notoriously unsatisfactory. The general term does not indicate that this is not a single pathologic entity but a group of etiologically different conditions. On the other hand, clear-cut classification is difficult for several reasons, one of which is the practical one that the various forms tend to merge into each other: an otomycotic infection, for instance, soon becomes bacterial also, as does a primary traumatic or hemorrhagic lesion. An acute infection may become chronic, and a chronic or subacute infection may present acute exacerbations. The American Academy of Ophthalmology and Otolaryngology or the American Otological Society would perform a useful service if an acceptable classification could be devised and generally adopted.

MANAGEMENT OF SPECIAL TYPES.

Circumscribed Otitis Externa. Circumscribed external otitis differs in no respect except location from a furuncle elsewhere in the body, and in the absence of treatment its natural history is the same. In the occasional case it may be possible to abort it by the use of small doses of X-ray. Usually it is seen later, and treatment must begin with the relief of pain by diathermy, external heat, analgesics, and small doses of X-ray, according to indications. Wicks saturated with iodex ointment are inserted into the ear if the epithelium is grossly intact; otherwise, cresatin-soaked wicks are used. Hot compresses may be employed to expedite localization, which is the objective of all treatment. It is sometimes wise to remove the tops of localized lesions, but surgical incision and drainage are practically never necessary.

Recurrent furunculosis demands a search for, and elimination of, possible foci of infection. Diabetes should always be excluded as a possible cause. X-ray applications, three times weekly in 100 r. dosages, to a total dosage of 500 r., will control some cases. Staphylococcic toxoid and autogenous vaccines may be tried. If the condition is obstinate it is well to resort at once to one or two intramuscular injections of penicillin (400,000 units).

If furunculosis of the external ear is secondary to otitis externa diffusa or otitis media, the patient should be hospitalized and treated with penicillin (400,000 units) every 12 hours together with streptomycin (0.5 gm.) every six hours, the reason for the combination being that these lesions are likely to be Gram-negative as well as Gram-positive.

Diffuse Otitis Externa. By the trial and error method I have come to limit the agents I usually employ in otitis externa diffusa to sulfamylon for Pseudomonas and other Gram-negative infections, aureomycin for Staphylococcus and other Gram-positive infections, and cresatin, with or without thymol, for fungoid infections. Since I have followed this plan I have had almost uniformly good results, and I recommend it to you as being both simple and logical.

Sulfamylon, my own laboratory studies[19] show, is consistently effective *in vitro* against the organisms commonly found in otitis externa, solutions varying in strength from 4 per cent down to 0.25 per cent effectively inhibiting growth in all the cultures tested. Additional studies showed that the optimum bactericidal effect was obtained when a 1 per cent solution was kept in contact with the bacteria for a five-minute period, about as long, incidentally, as a patient who is not incapacitated by his disease will submit to inactivity at any single time. Although streptomycin would probably be as effective against the bacteria susceptible to sulfamylon, the latter is preferred because its pH is acid, while that of streptomycin is alkaline.

At the patient's initial visit, after careful cleansing, the fungicide of choice (cresatin, with or without thymol) is applied if the disease is thought to be fungous in origin, which most of the time it is not. Otherwise, sulfamylon is applied and the patient is shown exactly how to use it. He lies on the nonaffected side, instills the 1 per cent aqueous solution into the ear, and permits it to remain for exactly five minutes. The ear is then blotted dry with cotton and a cotton wick is inserted, which is changed often enough to keep it from being saturated with discharge. Treatment is carried out twice a day.

If severe cellulitis is associated with diffuse otitis externa it is well to delay sulfamylon therapy for 24 hours and in the interim employ wicks saturated with Burow's solution. They are kept moist by the application of drops for 15 minutes every two to three hours. Dry cotton wicks are used between treatments.

When the patient returns for his second visit, usually 48 hours later, the further procedure depends upon his progress. The ear, as a rule, is greatly improved, probably as the result of the thorough cleansing as well as of the action of sulfamylon. If the infection is clearly under control the ear is again cleansed, then is dried thoroughly and dusted with sulfanilamide powder. The patient is instructed to discontinue all medication and to make every effort to keep water out of the ear for the next three to four weeks. If the response has been less than satisfactory, the initial treatment is repeated, and the patient returns for further treatment and observation, preferably every 48 hours, until control is achieved.

If good results are not apparent at the second visit, smears and cultures are made and subsequent therapy depends upon the laboratory findings. The report usually shows *1.* mixed organisms with Pseudomonas predominating, *2.* mixed organisms with Staphylococcus predominating, *3.* Streptococcus with the precise type named, or *4.* B. pyocyaneus. If a pure Staphylococcus is reported, or a mixed infection with Staphylococcus predominating, a coagulase test is run. If it is reported negative, the Staphylococcus is disregarded and sulfamylon is continued, as if the infection were Gram-negative. If the test is reported positive, sulfamylon therapy is discontinued and the patient is treated with aureomycin, in the form of ophthalmic solution. I find this method far more effective than the penicillin therapy (intramuscular and parenteral) which I formerly used.

A precaution which should not be omitted after the conclusion of treatment is the healing of any fissures of the meatus which may be present. They are cleansed, cauterized with 10 per cent silver nitrate, and then dusted with sulfanilamide powder.

Otomycosis. If the otitis is thought to be fungous in origin, 1 per cent thymol in cresatin solution is applied to the external canal following cleansing, which must include the removal of all visible scales. A wick saturated with the same solution is then left in place for 24 hours. Blonds, or patients whose skin seems sensitive, are treated with a dilute solution of cresatin (50 per cent) and usually without thymol. After the wick is removed, the solution is continued as drops, applied in the usual manner, twice daily.

The point of this therapy is the keratolytic action of cresatin. Cresatin alone, or thymol alone, or the two drugs in combination, were the most effective tested by McBurney and Searcy[1] against Aspergillus, and in the years that have passed since their investigation no more effective fungicides have been found. If the thymol is too irritating, cresatin alone seems equally useful. In the McBurney-Searcy studies the *difference in their in vitro effectiveness was only fractional.*

Senturia's[9] studies on the fungi most often found in the ear showed that sulfanilamide is markedly effective against aspergillus, penicillium and mucor though not against monilia. He, therefore, recommended a powder composed of sulfanilamide for its fungicidal action, sulfathiazole for its antistaphylococcic action, and zinc peroxide for its oxidizing effect in the proportions 4:4:2. Like Senturia, my experience with this powder has been that, when it is applied, pain is promptly relieved or greatly diminished, edema is decreased, and the discharge becomes much less. The promptness of results is proportionate to the severity of the case. I have found, however, that this is not a stable preparation, and I am beginning to use sulfanilamide powder alone, or the Whalen preparation containing thymol, after cresatin-thymol therapy is discontinued.

Eczematoid Otitis Externa. With the measures just described, about 75 per cent of all patients with external otitis will get well promptly or with reasonable promptness. The remaining 25 per cent furnish most of the problems.

The type of external otitis loosely classified as eczematoid heads the list of these problems. The infections are frequently of low grade and of great chronicity. They require intensive therapy, which must be continued over long periods of time. Often it is necessary to substitute one drug for another. Treatment is frequently complicated by the fact that the patient has had the disease for a long time and has had many previous forms of treatment.

In this type of case the initial investigation is thorough. The general as well as the local condition is surveyed, and such consultations as are necessary are secured. Laboratory investigations include cultures, smears, blood serology, complete blood picture, and hematocrit. Foci of infection are searched for. Allergic possibilities are investigated. Nothing, in short, is omitted which might throw light upon the picture. All medication is discontinued and treatment is limited to simple cleansing and the use of dry wicks until the etiology is established. Cosmetics are discontinued and nonallergic products are substituted. The patient is warned against scratching the ears, and gloves are recommended for night wear to prevent it. A possible psychogenic origin is always suspected in this type of case. My own experience, in fact, indicates that this type of persistent otitis externa does not occur in stable persons.

It is difficult to generalize about the treatment of eczematoid otitis externa, since it depends chiefly upon the underlying cause. It may include the vitamin therapy, especially thiamin chloride, liver extract or thyroid extract. If the skin has become hyperkeratotic, some keratolytic agent should be used briefly, usually some preparation of salicylic ointment. If secondary infection has occurred, aureomycin is preferable to penicillin, since eczema occurs only in hypersensitive patients, who are frequently sensitive to the later drug. Radiotherapy in small doses may be useful. It is difficult to evaluate the forms of therapy suggested in some contributions to the subject since the variety of agents used simultaneously make it impossible to determine the worth of any single drug.

Other Types. The various forms of true allergic otitis externa, with which the eczematoid variety overlaps, are readily treated by removal of the causative agent, followed by strict cleanliness and the application of some soothing ointment. It is my experience that in many instances allergic otitis is induced and perpetuated by the therapy used to treat otitis externa.

Seborrheic dermatitis, which is not a very common variety, can be treated only in combination with treatment of the scalp disease, which is usually primary. Treatment consists of the use of a keratolytic agent followed by a bland ointment, to restore the skin to its normal condition.

Erysipelas, which is a streptococcic infection, is best treated in the ear, as elsewhere on the body, by a combination of sulfathiazole by mouth and penicillin intramuscularly. Traumatic external otitis requires little more than the elimination of the traumatizing agent, after which natural healing is permitted to occur. If infection is superimposed, the treatment is that used in bacterial otitis externa.

In granulomatous otitis externa, gentle, daily cleansing is the most important part of therapy. Pedunculated granulations may be removed with forceps, or may be touched with a probe dipped in silver nitrate or pure carbolic acid. The ear is then dusted with sulfanilamide and dry wicks are applied by the patient as necessary. Healing is slow and new lesions may appear even after it seems to have occurred. In this type of otitis externa the organisms present are usually those which normally inhabit the external auditory canal and, therefore, the use of antibiotic agents is unnecessary. It is usually wise, however, to run a cytologic test, to check for mucus threads, whose presence would indicate an overlooked pin-point perforation of the tympanum.

Hemorrhagic otitis externa (bullosa hemorrhagica) is best treated by mechanical rupture of the bullae, under strict aseptic technique, with a myringotome or some similar knife. Pain is usually promptly relieved. Bonain's mixture is applied for 15 minutes before the incision of the lesions, to anesthetize the parts.

Chondrodermatitis, which is a very painful and quite uncommon condition, responds only to some method of cauterization or complete excision. Chemotherapy and antibiotic therapy are always indicated in perichondritis.

A wide variety of purely dermatologic conditions may affect the ear, and a consultation with the dermatologist is often indicated. The otologist, however, must continue to control therapy, regardless of the consultant called into the case, since treatment of the external auditory canal is always indicated and no other specialist is trained or equipped to apply it.

Up to very recently the treatment of herpes zoster oticus was entirely symptomatic and not at all satisfactory. Recently, on the suggestion of Hilger,[20] I have been using intravenous injections of procaine, ascorbic acid and adrenalin, the precise method depending upon the type of case. Aureomycin is used with this regimen, and the results, in the few cases I have treated by this means, far surpass the results of any other method I have previously used.

THE NEWER DRUGS.

I regret that because of lack of time I cannot discuss the newer drugs recommended for otitis externa. I have not duplicated the good results reported by Reardon[21] and by Walker[22] with isopar, chiefly because my patients have shown such a degree of sensitivity that I could not continue its use. I have had much the same experience with furacin as well as with compound 280 (dibromsalicyladehyde), with which Hayes and Hall[23] have had a favorable experience. Gill's[2] objection to the latter preparation on the ground of its high pH (8.0 to 8.2), which is definitely on the alkaline side, is well taken. Sullivan and Smith,[24] because so many of their patients were sensitive to it, now use it only as a last resort. The excellent results reported by Ochs[25] from acetic acid, which he used in the form of household vinegar, are so impressive that the method seems well worth a trial. Nelson's[26] results in the Pacific were equally good. The "sloppiest ears," he reported, were often clean, dry and healthy-looking 48 hours after treatment was begun, though he warned that recurrence was likely if applications were not continued for several days longer.

PROPHYLAXIS.

Let me conclude this presentation with a few words about prophylaxis in otitis externa. I doubt that any of the various methods which have been suggested is as effective as the simple plan of keeping the ears clean by the ordinary rules of cleanliness, rinsing them free of soap, drying them carefully whenever they have been wet, and then letting them entirely alone. This prohibition extends to the removal of wax, which should be left undisturbed unless for any reason an otologist thinks it necessary to remove it.

Certain predisposing factors cannot be controlled, such as the temperature and humidity in tropical climates, but special care can be exercised to keep the ears dry when perspiration is profuse. Even those who do not accept swimming as a usual cause of otitis externa — as I do not — would agree that it is well to avoid this exercise during the course of the disease and until treatment is concluded. Some observers believe that methods intended to protect the ear during bathing and swimming actually predispose to infection. My own feeling is that if any such attempt is made it should be limited to the use of lamb's wool plugs. If mothers insist on something more impressive, they may be given a simple solution of mercuric chloride (1:5,000) in 50 per cent alcohol, to be used as drops, after which the ear is blotted dry.

The continued use of alcohol, salicylic acid, boric acid, acetone, sulfanilamide powder and other agents in various combinations has been recommended, but I think the current opinion is that the ears are best left alone except for the simple hygienic measures already mentioned, on the ground that the more closely normal physiologic conditions are approximated, the less likely is a recurrence of infection.

SUMMARY.

The treatment of otitis externa should be a matter of principles rather than of the use of special agents. Once principles are established and understood, other therapeutic details fall into their proper relationship. The best results are secured by otologists who limit themselves to a few agents, select them because of their antibacterial or fungicidal effects, employ them for as brief a period as the necessities of the case permit, and supplement them by thorough cleansing of the external auditory canal and by measures for the relief of discomfort.

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A NEW INSTRUMENT FOR USE IN MAKING ANTRUM WINDOWS.*

MORRIS DAVIDSON, M.D.,
St. Louis, Mo.

Otolaryngologists who have made antrum windows are aware of the many difficulties that accompany such procedures. Several different instruments must be used and frequently these instruments do not possess the necessary angle or curve to permit satisfactory removal of the bony tissues in the lateral nasal wall.

An antrum window perforator and enlarging cutter of one piece has been devised which eliminates the necessity for varied perforating, rasping and biting instruments. This instrument has a chain drive and can be quickly attached to any standard dental handpiece. The dental engine should be operated at the lowest speed. The angle of the tip is approximately 45 degrees. Employing the dental engine and handpiece which are now available in most operating rooms, the entire procedure of making antrum windows can be accomplished with this single instrument.

The first portion of the instrument is a perforator burr for use in entering the maxillary antrum through the inferior meatus. The portion behind the perforator is a concave cutter. When the perforation has been accomplished the instrument is moved in circular manner and the concave cutter portion is used to enlarge the window to the desired size. In a matter of a few seconds a large antrum window can be made with smooth edges, the margins of which can be extended in any direction with ease.

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*From the Department of Otolaryngology, Washington University Medical School, St. Louis, Mo.

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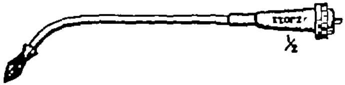
This instrument has also been used to enter and enlarge the opening through the canine fossa during the Caldwell-Luc operation.



Because of the possibility of explosion, this instrument has not been used in those cases operated under general anesthesia in which one of the explosive anesthetics has been used.

KENFIELD MEMORIAL SCHOLARSHIP.

In 1937 a sum of money was subscribed in memory of Miss Coralie N. Kenfield, of San Francisco, Calif., a teacher of advanced methods in teaching lipreading. This money, placed in the Kenfield Memorial Fund, is administered by the American Hearing Society and provides an annual scholarship. The amount of the Kenfield Memorial Scholarship for 1951 is $100.00.

Applications for the scholarship will be considered from any resident of the United States who desires to teach lipreading (speechreading) with or without other types of hearing and speech therapy, and who can meet the following requirements:

A. Personal. Well adjusted individual with a pleasing personality, legible lips, a good speech pattern and no unpleasant mannerisms. *B. Education.* College graduate with a major in education, psychology, and/or speech. If the applicant is hard of hearing, 30 clock hours of private instruction under an approved teacher of lipreading or 60 clock hours of instruction in public school classes under an approved teacher of lipreading are required.

The winner of the scholarship may take the Teacher Training Course from any normal training teacher or school or university in the United States offering a course acceptable to the Teachers' Committee of the American Hearing Society. The scholarship must be used within one year from the date the award is made.

Applicants must be prospective teachers of lipreading to the hard of hearing. Those already teaching lipreading cannot be considered.

Applications must be filed between March 1 and May 1, 1951, with: Miss Rose V. Feilbach, Chairman, Teachers' Committee, 1157 North Columbus Street, Arlington, Va.

January 1, 1951.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

As of April 1, 1950.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-4 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1; Otarion Model E-1S; Otarion Model E-2; tarion Model E-4; Otarion Models F-1 and F-2.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable)—*Jour. A. M. A.,* 109:585 (Aug. 21), 1937.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid—*Jour. A. M. A.,* 139:785-786 (Mar. 19), 1949.

Manufacturer: Precision Electronics Co., 850 West Oakdale Ave., Chicago 14, Ill.

Sonotone Professional Table Set Model 50—*Jour. A. M. A.,* 141:658 (Nov. 15), 1949.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Meeting: Havana, Cuba, 1952. For information, address Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

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Meeting: White Sulphur Springs, W. Va., May 7-8, 1951, P. M.

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Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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EMBRYONIC HEARING ORGANS AFTER MATERNAL RUBELLA.*†

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Since the first communication of Gregg, in 1941, on congenital defects in the progeny after rubella (German measles) of the mother, suffered before or during pregnancy, the question has been discussed with increasing momentum. The present paper is restricted to one detail: deafness in the progeny. As to survey of the literature, at least two competent studies give discussion and extensive bibliographies up to 1949: the thesis of Candiotti, from the Department Portmann, in Bordeaux, and the thesis of Kamerbeek, from the Department Van Gilse, in Leiden.

So far many aspects remain unsettled, even such basic ones as frequency of occurrence. Hiller examined 42 congenitally deaf children in the Tasmanian Institute for the Blind and Deaf. In 32 cases the deafness was due to rubella of the mother in the early months of pregnancy. The remaining 10 were classed as nonrubella inner ear deafness cases, but there

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was reason to believe that a proportion of these may have resulted similarly from rubella infection of the mother. On the other hand, the Massachusetts survey of Ober, Horton and Feemster found only one case of deafness among the progeny of 54 mothers with rubella during their pregnancies. In this single case the German measles were contracted in the third month.

The situation was summed up by Fowler, in 1950, when he wrote that almost 100 per cent of the offspring have been reported to have congenital defects if rubella was acquired during the middle or first trimester of pregnancy.

The deafness itself is described as of varying degrees up to a complete one with no regular type or curve, even right and left in the same child differing considerably.

In spite of the great amount of published material, pathohistological investigations are conspicuously scarce.

MATERIALS AND METHODS.

The basis of this report is a study of serial sections of three temporal bones originating from two embryos removed by elective abortion after the mothers suffered rubella in the first months of pregnancy. For the autopsy material we are indebted to A. T. Hertig, Professor of Pathology at the Boston Lying-In Hospital.

Case 1: The mother contracted rubella in the eighth week of her pregnancy; in the fourth month the embryo was removed by operation.

One of the temporal bones was secured and after the usual histological preparation sectioned in a roughly horizontal plane. The staining applied was Masson's and a number of sections underwent Bodian impregnation.

The microscopical examination revealed the following: The most conspicuous feature was given by hemorrhages, mostly in the external and the internal ear. In and around the external ear engorgement and extravasates were seen in the temporal muscle, in the skin and in the cartilages of the auricle (see Fig. 1). Hemorrhage had separated the inner lining of the tympanic membrane in circumspect areas, forming cyst-like spaces inside the membrane and between the membrane and the mesenchyma filling the middle ear; in the latter, pneumatization was initiated in several areas.

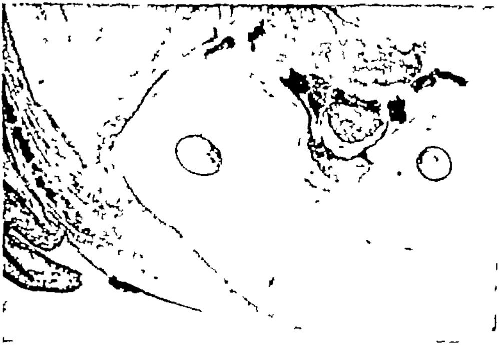

Fig 1 Massive hemorrhage at the bottom of the subarcuate fossa. Masson, ×17

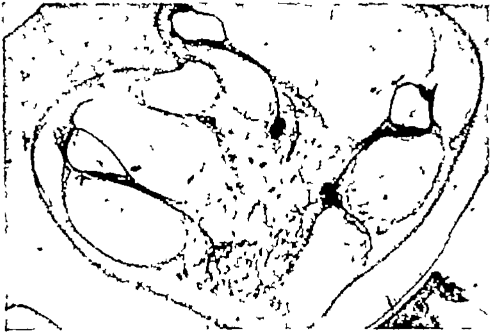

Fig 2 Cochlear ramuli of the acoustic nerve with spiral ganglion in the connective tissue modiolus of the cartilaginous cochlear capsule. Bodian impregnation, ×28

Around the inner ear free blood occupied the bottom of the subarcuate fossa (see Fig 1) as well as the internal acoustic meatus In the latter, massive bleeding enveloped the cochlear and vestibular branches, while the cochlear branch was damaged by intraneural hemorrhage as well Inside the cochlea the cochlear artery, in the axis of the connective tissue

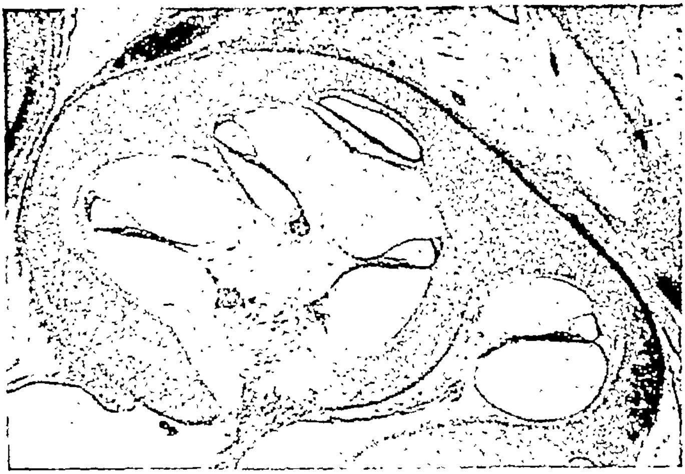

Fig. 3. Spiral ganglion and trunks of the cochlear nerve in the connective tissue modiolus of the cartilaginous cochlear capsule. Hematoxylin-eosin. ×28.

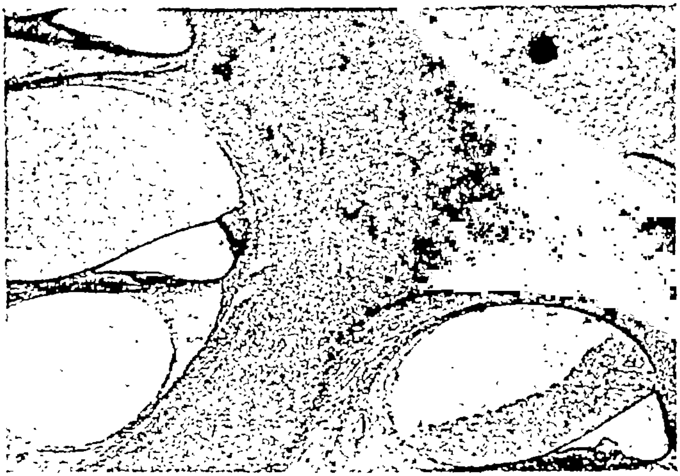

Fig. 4. Corti's organ in the cartilaginous cochlear capsule, beginning to be differentiated in the apical turn; well demarcated in the medial turn; much farther advanced in the basal turn. Hematoxylin-eosin, ×62.

modiolus, was engorged. Blood was seen in all of the three scales. Reissner's membrane was bulging in all turns, with the highest degree of dilatation in the apical turn (see Figs. 4 and 5). Corti's organ was

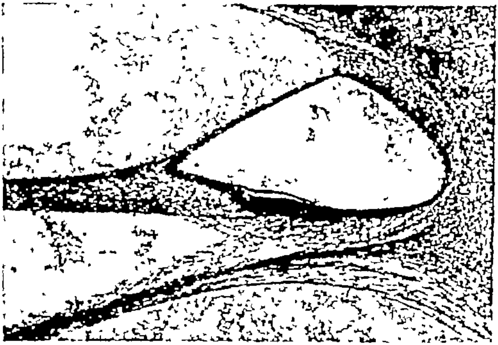

Fig. 5 Corti's organ in an apical turn, flat row of cells giving the impression of a pseudostratified epithelium, covered by the tectorial membrane. Hematoxylin-eosin, ×145.

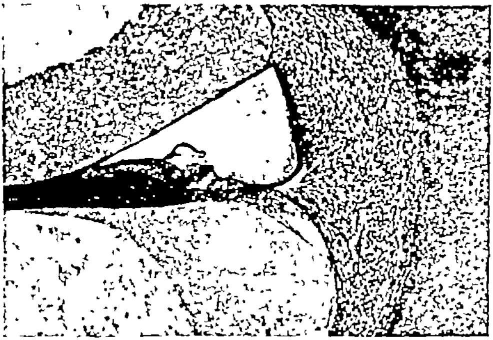

Fig 6 Corti's organ in a basal turn: loosened cell groups, bulging Hematoxylin-eosin, ×145.

represented in the middle and in the apical turn (see Figs. 4 and 5) merely by a simple row of cells with the membrana tectoria stretched flatly over them. In the basal turn, Corti's organ was more advanced as to differentiation (see Fig. 6), the flat row being loosened and elevated into a mound with the tunnel clearly discernible. The semicircular canals showed scattered hemorrhages in the endo- as well as in the perilymphatic spaces, in some points even in the endosteum of a semicircular canal.

Case 2: The 25-year-old primipara contracted rubella six and one-half weeks after conception. As the data of the latter was known, it was possible to compute exactly the age of the embryo, which was removed on the 109th day (at 15 and one-half weeks) by elective abortion. The crown-rump length was 118 mm. Microscopical examination of the eyes (Dr. D. G. Cogan) showed no cataract. The examination of the temporal bones gave the following results:

Right Temporal Bone: Cut in a vertical plane, the pilot series was alternately stained with hematoxylin-eosin and left unstained for observation in polarized light. Engorgement of the soft tissues was conspicuous all over this temporal bone, with hemorrhages scattered about the middle and in the internal ear. In the hypotympanum they appeared in more or less massive form along the walls. They were seen around the crura of the stapes. Between the dura and the labyrinthine capsule, and at the bottom of the subarcuate fossa slight bleedings were found. Pneumatization started in the middle ear, with an open space formed along the tympanic membrane. The vestibular and tympanic scalae were filled with mesenchyma, with the cochlear duct entirely free everywhere. Reissner's membrane was slightly bulging. Corti's organ was represented in the apical turn by a simple row of cells, while in the middle and in the basal turn its development advanced farther, showing a slender mound with the tunnel discernible.

Left Temporal Bone: This was unfortunately removed by a cut which went straight through the cochlea. To avoid further loss of material this accidental plane was maintained under the microtome, with the result of an approximately vertical series of sections. The sections of the pilot series were alternately stained with hematoxylin-eosin and left unstained for observation in polarized light. Beginning pneumatization was seen in the middle ear. In the cochlea, fibrinous masses filled the vestibular and the tympanic scala, while the cochlear duct was free. Corti's organ showed a more progressed stage in the basal than in the middle and in the apical turn. The cochlear aqueduct was filled with a fine meshwork of fibrillary nature. In the semicircular canals, the endolymphatic canals were free while mesenchyma filled the perilymphatic spaces. Opposite the entrance to the modiolus, the acousticofacial ganglionic complex was seen in the adequate stage of development.

COMMENT.

The histological findings have shown features of interest which can be divided into a traumatic and a developmental group.

Two of the temporal bones contained hemorrhages in their different parts besides engorgement of the vessels in the soft parts (see Fig. 1). Seen in the tissues and cavities of the external, middle and internal ear they must be considered a result of operative trauma. In one temporal bone, engorgement was seen distinctly in the cochlear artery, running in the axis of the connective tissue modiolus.

In the scala vestibuli of an embryo originating from a rubella-mother, Nauta (quoted by van Gilse) saw peculiar blood cell formations of probably pathological significance. van Gilse and Kamerbeek observed hemorrhage in the vestibular scala of a child, deaf from rubella of the mother. De Roetth and Greene reported bloody stools in the newborn child of a mother who had rubella in the fifth week of her pregnancy.

The bleeding tendency in rubella manifests itself in several ways. Wesselhoeft enumerated reports on thrombopenic purpura in connection with rubella. Vascular drainage resulting in thrombotic occlusion through disturbed clotting mechanism was claimed by Putnam to underly encephalitis and meningeal irritation in rubella. Adler's tabulation of 100 encephalitis and encephalomyelitis patients from the Department of Neurology, Harvard Medical School, contained three cases following rubella with no death — and no deafness; they represented "proliferative" encephalitis (proliferation of the glia cells and other tissue elements); the author quoted Ford's case which suggested that toxic as well as proliferative lesions might be present.

It is impossible to decide whether the rubella infection by itself is able to produce hemorrhages, although such a possibility cannot be denied. The difficulty in evaluating hemorrhages may be illustrated by the example of congenital syphilis and by the discussion going on since the publications of Baratoux in 1886-1887, as to whether they are part of the basic pathology or not. Mosher pointed out that hemorrhage in the cochlea is rarely if ever an artefact of a similar character; he wondered whether hemorrhages might not leave behind fibrous tissue in embarrassing locations. Alexander wrote on the danger of excessive scepticism in judging pathological pictures of this kind. The significance of perineural and intraneural interstitial hemorrhages within the internal acoustic meatus and in the intratemporal channels has been discussed previously; and it was found that cystic "loculaments" of traumatic origin or following hemorrhages in general (see Fig. 1), are sources of cholesteatomas in extrauterine life (Kelemen[1,2]).

In the present specimens, the cochlear aqueduct was closed by a network of mesenchymal tissue, and in this way, even with hemorrhages in the subarcuate fossa and other subdural locations, there was no possibility for invasion of the intracochlear space by this channel.

Hemorrhages as signs of damage done to the vessels (shown in Fig. 1) make one anticipate trouble at the time of the delivery. At this period increased vascular vulnerability will produce pictures which belong to the realm of birth trauma.

The degree of development was found to correspond to the embryonic period. The annulus stapedius, of visceral origin, was firmly fused with the lamina stapedialis, of capsular origin, with beginning differentiation of the annular ligament. The otic capsule presented the homogeneous texture of the original cartilage, with a few areas of ossification disseminated along the semicircular canals. No traces of ossification were found in the connective tissue modiolus; intramembranous bone is first deposited here, according to Bast and Anson, at the fetal age of 20 to 21 weeks. The vestibular end-organs showed the appropriate stage of development. With mesenchyma filling almost completely the vestibular and the tympanic scalae the lumen of the cochlear duct was free; this is in accord with the fact that it differentiates earlier than the perilymphatic scalae.

The organ of Corti followed, in the three turns, the rule that differentiation advances progressively toward the apex (see Figs. 4, 5 and 6). The simple row of cells as seen in the apical turn, reminiscent of pseudostratified epithelium, underwent in the basal turn the characteristic loosening, with appearance of the tunnel and a bulging of the whole cell group, on the way to the definitive formation of the papilla. It is known that an actively differentiating cell is more vulnerable *than a cell in a pre- or in a postdifferentiation stage.* The ramuli nervi cochlearis between papilla and spiral ganglion were clearly outlined, with the ganglion formed according to the fetal stage. Among the fibres in the axis, converging to form the trunk of the cochlear nerve, those coming from the basal turn were the most massive (see Figs. 2 and 3).

At this point the findings of Carruthers are of interest. He examined the temporal bones of an infant who died at the age of six and one-half months. The mother suffered from *rubella during the first month of pregnancy.* The infant had congenital cataracts, patent ductus arteriosus and was thought to be deaf. The semicircular canals, bony and membranous, were well formed. The outstanding feature was the total absence, in both ears, of any differentiation of the primitive cells to form the organ of Corti. The vascular stria contained relatively few vessels, and was shallower and less cellular than usual.

Regarding the mechanism of the effect of a possible toxic agent as to its topographical sequence, observations on animals with spontaneous or experimental cochlear damage give information. In the waltzing guinea pig (Lurie[1]) the degeneration of the organ of Corti, an inherited characteristic, starts in the first and second turns, and progresses towards the helicotrema. It is not a gradual, progressive degeneration, but an irregular one. A great number of the animals examined showed a small remnant of organ of Corti nearly *normal at the helicotrema. The original organ of Corti is* finally replaced by a simple cuboidal epithelium. The spiral ganglia showed an amount of degeneration which was less than that expected with complete degeneration of the organ of Corti. Nerve fibres were still going to the basilar membrane with no organ of Corti present. This again indicates, as shown in the normal development of the cochlea (see Figs. 2-6), that the organ of Corti is not bound up as intimately with its nerve supply as has been thought.

In animals exposed experimentally to a mechanical or acoustic trauma it was found (Lurie[2]) that disintegration of the cells of the organ of Corti takes place with toxic chemical changes in the scala media and a possible spreading of the degeneration of the organ of Corti throughout the cochlea; this is a condition progressing for long periods after the actual damage was suffered. In the same species (the guinea pig), the organ of Corti was remarkably resistant amid the changes of spontaneous serous labyrinthitis (Kelemen[3]). Rüedi and Furrer discussed in detail the course of pathological changes in the end-organ and in the nervous apparatus, including their interrelation, in acoustic trauma.

Ingalls, discussing the stage-specificity of this first-trimester infection, mentioned arrested development and vascular anomalies among the possible causes. His Table 1 showed the mean critical period for the development of deafness at 2.17 months of prenatal life. Dietary deficiencies, toxins, duration of stay in oxygen of the premature baby, anoxia by obstetric sedation and anesthesia, may enter the picture in different combinations.

TABLE 1.

SUMMARY OF MICROSCOPICAL FINDINGS IN THE TEMPORAL BONES.

Hemorrhages:

- External ear
  - in skin, temporal muscle, auricular cartilages
    - engorgement, extravasates
- Tympanic membrane
  - in cyst formation originated by separation of the inner layer
- Middle ear
  - hypotympanum
    - along walls
  - around crura of the stapes
- Internal ear
  - outside otic capsule
    - at bottom of the subarcuate fossa
    - in internal acoustic meatus
      - perineural
        - in cochlear branch
        - in vestibular branch
      - intraneural
        - in cochlear wall
  - inside otic capsule
  - in cochlea
      - cochlear artery, in axis of modiolus, engorged free blood in the three scalae.
    - semicircular canals
      - scattered in endo- and perilymph spaces

Developmental Stage:

- Mesenchyma
  - Middle ear
    - pneumatization starts along tympanic membrane
  - Internal ear
    - endolymph spaces free
    - perilymph spaces filled
    - cochlear aqueduct filled by fine meshwork
- *Internal ear*
  - Reissner's membrane
    - slight bulging in almost every turn
  - Corti's organ
    - apical turn: row of cells like pseudostratified epithelium, tectorial membrane stretched out flatly over them
    - middle turn: in one temporal bone as in apical turn; in the others further advanced in differentiation
    - basal turn: row of cells loosened, elevated into a mound, tunnel discernible
  - Nervous apparatus
    - trunk, spiral ganglion and ramuli of the cochlear nerve in the connective tissue modiolus of the cartilaginous cochlear capsule well developed
- Acousticofacial ganglionic complex
  - in adequate stage of development

Hopkins found no direct correlation between the degree of deafness and the month of maternal rubella, with many cases of profound deafness after infection in the second and third month and with cases of partial deafness in the first month of pregnancy. This contrasted with the experience of Lewis at the Winthrop Foundation at the Massachusetts Eye and Ear Infirmary who found generally a more profound deafness with early maternal infection.

A parallel with the deafness as found by Goodhill in erythroblastosis fetalis shows points of contact as to the possible etiology, as the factor of anoxia, and as to clinical significance. Here as there, among the genetic factors, the father has to be considered. Incidence in the first as against incidence in the subsequent pregnancies has to be evaluated. Audiometric symmetry, both for frequency and intensity, can offer help in finding out about the localization of the damage.

Fox, Krumbiegel and Teresi examined congenital anomalies after maternal measles, mumps and chicken pox. In all cases of deafness a conduction deafness from otitis media was found, none showing evidence of congenital origin.

Considering the irregularity of findings regarding the hearing *of children after rubella of the mother (islands of hearing)*, the difference in the developmental stage of Corti's organ along the basal lamina is worth watching. The progress between the basal and the apical turns might be interrupted in this period of delicate adjustment, beyond which, according to the general consent, the danger of later deleterious consequences seems to be mostly gone. The lagging behind of one section would produce unpredictable damage to the hearing. A sufficiently large material is needed to obtain information about this important point. van Gilse emphasized that real progress can be expected only when it will be possible to experiment with the pathological agent itself. In all this it has to be considered that, according to Baumgartner, congenital malformation and injury at birth were found, for 1947, among the five leading causes of infant death in the United States.

SUMMARY.

Three temporal bones of two four-month embryos, operatively removed from mothers who suffered rubella in the second month of pregnancy, were histologically examined. Hyperemia and free hemorrhages were seen, while the nervous elements of the inner ear were found to be in a condition corresponding to the fetal age.

This being the period in which the organ of Corti is differentiating along the basilar membrane, any possible disturbance in the delicate mechanism of adjustment, *e.g.*, the lagging behind of certain sections, has to be closely watched as a possible source of hearing defects.

The specific infectious agent causes arrest of development of the end-organ in the most critical period and vascular damage, possibly the latter being the primary change. Vulnerability of the vessels shows up at the end of the pregnancy

in an increased tendency for birth injuries This way a series of factors collaborate to produce the picture of defective hearing in the progeny, after rubella of the mother

As far as a survey can be given today, there are two ways in which damage results by exposure to the virus of rubella one being arrest of development, the other vascular changes The two can be formulated into a monistic theory by assuming that the vessels are the first to suffer, and lack in nutrition is then followed by developmental arrest Here is a field of research of rare interest with many problems unsolved The first decade after the basic discovery of Gregg yielded a rich crop of investigative results, among which, however, pathological findings are negligible. Aycock said that rubella now takes on the status of a disease worthy of the best in diagnosis, treatment, prevention and research In attacking the problem of the pathology of aural damage it is desirable that the otologist should assume the leading rôle before the initiative is definitely lost to borderline disciplines

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ACUTE LARYNGOTRACHEOBRONCHITIS. AN ANALYSIS OF 1,175 CASES WITH 98 TRACHEOTOMIES.*†

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There were 1,175 cases of acute obstructive infections of the larynx, trachea and bronchi treated on the croup service at Willard Parker Hospital during the past decade, 1940-1949 inclusive. Of these, 98 (8.3 per cent) were diagnosed as being of diphtheritic origin, and 1,077 (91.6 per cent) nondiphtheritic. At this same hospital the incidence of diphtheria, in a series of 344 croup cases reported by Tolle,[1] in 1929, was over 60 per cent. In 1948, the incidence of croup caused by diphtheria was only 5 per cent (see Table 1). Last year there was only one case.

Of the total 1,175 cases, 32 died, making a total mortality rate of 2.7 per cent. There were 11 diphtheritic croup deaths with a mortality of 11.2 per cent, which was little changed from the 12 per cent reported by Neffson and Wishik in their series of 261 cases treated at Willard Parker Hospital in years 1931-1934;[2] however, there was a dramatic lowering of the mortality rate to 2 per cent in the nondiphtheritic acute obstructive cases as compared with a similar group of 1,379 cases during the preceding decade, 1930-1939, in which there were 136 deaths with a mortality rate of 9.8 per cent (see Table 2).

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*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, Nov. 15, 1950.

†Thanks are due Mr. Charles Petersen for helpful assistance in the art work used in this paper.

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Table 1.

TOTAL OBSTRUCTIVE CROUP CASES AT WILLARD PARKER HOSPITAL
FOR THE 10-YEAR PERIOD 1940-1949

YEAR	CROUP	CASES	DEATHS	% MORTALITY	LARYNGOSCOPED	SUCTIONED	TRACHEOTOMIES	DEATHS	% MORTALITY
1940	DIPHTHERITIC	14	1	7.1	14	14	4	1	25.0
	NON DIPHTHERITIC	155	8	5.1	137	68	24	5	20.8
1941	DIPHTHERITIC	11	1	9.0	11	9	4	1	25.0
	NON DIPHTHERITIC	99	3	3.0	87	8	13	2	7.7
1942	DIPHTHERITIC	12	1	8.3	6	4	1	0	0
	NON DIPHTHERITIC	140	2	1.4	124	16	12	0	0
1943	DIPHTHERITIC	6	1	16.6	5	3	0	0	0
	NON DIPHTHERITIC	155	2	1.3	78	13	13	2	15.4
1944	DIPHTHERITIC	3	0	0	3	1	0	0	0
	NON DIPHTHERITIC	92	1	1.1	23	6	6	0	0
1945	DIPHTHERITIC	8	3	37.5	5	5	1	0	0
	NON DIPHTHERITIC	89	1	1.12	15	9	7	1	14.2
1946	DIPHTHERITIC	15	2	13.3	9	0	0	0	0
	NON DIPHTHERITIC	71	2	2.8	8	5	3	0	0
1947	DIPHTHERITIC	15	1	6.6	5	4	2	0	0
	NON DIPHTHERITIC	136	1	.7	9	10	1	0	0
1948	DIPHTHERITIC	13	1	7.7	7	1	0	0	0
	NON DIPHTHERITIC	77	1	1.3	14	9	4	0	0
1949	DIPHTHERITIC	1	0	0	0	0	0	0	0
	NON DIPHTHERITIC	63	0	0	4	4	3	0	0
TOTALS	DIPHTHERITIC	98	11	11.2	65	49	12	2	16.6
	NON DIPHTHERITIC	1077	21	2.0	299	134	86	10	11.6
GRAND TOTALS		1175	32	2.7	364	183	98	12	12.2

A more striking difference is noted in a comparison of two series during the last five years[3] as shown in Table 3. It will be noted that the total mortality rate for these respective periods has dropped from 9 per cent to 1.12 per cent and that the mortality rate in the cases in which tracheotomy was performed was reduced from 28 per cent to 5.5 per cent.

The partial conquest of laryngotracheobronchitis from its original rate of 50 per cent and higher to the present low rate is one of the romances of medicine. The current, almost universal administration of the antibiotics and of the sulfonamides both before and after the patient reaches the hospital is probably the greatest single factor in this favorable trend. The early administration of these agents by the physician in the home has done much to decrease the number of admissions to the hospital and to decrease the severity of the cases that are admitted. Graebner[4] in a review of the records of Willard Parker Hospital for the years 1937 to 1940 indicated this early trend of chemotherapy. He compared a group of

Table 2

COMPARISON OF FREQUENCY AND MORTALITY FOR SURGICAL INTERVENTION IN NON DIPHTHERITIC ACUTE OBSTRUCTIVE LARYNGITIS.

	1930 1939	1940-1949
TOTAL CASES	1379	1077
TOTAL MORTALITY, %	9.8	2.8
TRACHEOTOMIES	106	86
TRACHEOTOMY MORTALITY RATE, %	38.6	11.6
FREQUENCY OF TRACHEOTOMIES %	7.7	8.0
CASES INTUBATED ONLY	130	31
MORTALITY RATE OF INTUBATIONS	46.9	9.7
TRACHEOTOMIES WITHOUT INTUBATION	30	41
TRACHEOTOMIES WITH INTUBATION	76	45
TOTAL CASES INTUBATED	206	76
FREQUENCY OF INTUBATION %	15.0	7.0
TOTAL TRACHEOTOMIES AND INTUBATIONS	236	117
FREQUENCY OF TRACHEOTOMIES AND INTUBATIONS, %	17.1	10.8
RESULTING PNEUMOTHORAX	13	9
RESULTING PNEUMOTHORAX, %	12.2	10.4

Table 3

NON DIPHTHERITIC CROUP CASES AT WILLARD PARKER HOSPITAL A COMPARISON

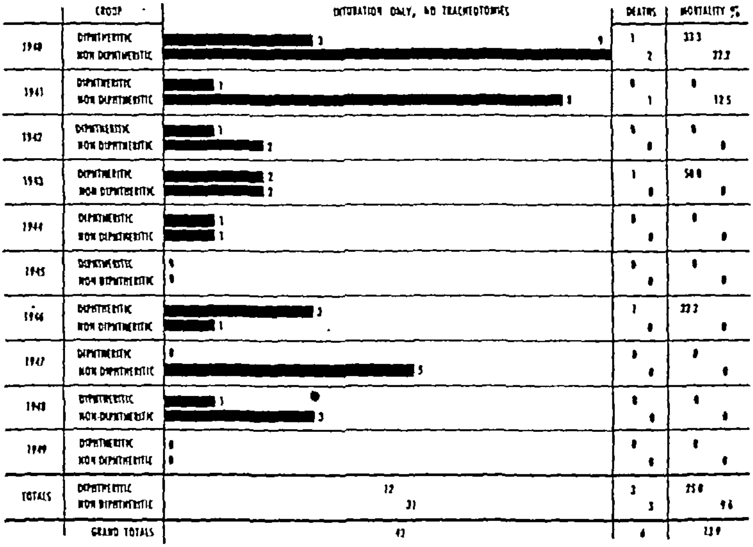
YEARS	TOTAL	DEATHS	MORTALITY %	TRACHEOTOMIES	DEATHS	MORTALITY %
[illegible]	414	36	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	5	[illegible]	[illegible]	1	[illegible]

217 patients admitted to the croup wards in the years 1937 and 1938 receiving no chemotherapy with a similar group of 241 admitted in 1939 and 1940 receiving chemotherapy. The

mortality rate of the later group was approximately one-third less. The major contribution of penicillin is indicated by comparing the three-year period, 1940-1942, when the sulfonamides were widely used with the three-year period, 1946-1948, when both the sulfonamides and penicillin were used. There was a further 50 per cent decrease in the mortality.

That there has been a definite and persistent trend away from mechanical and surgical intervention is clearly illustrated in Table 2. It will be seen that in the last decade the combined frequency of tracheotomy and intubation has decreased from 17.1 per cent to 10.8 per cent. Although the total frequency of tracheotomy increased slightly from 7.7 per cent to 8 per cent, there was more than a 50 per cent decrease in intubations with an increasing number of cases tracheotomized without previous intubation. This is not surprising as the present tendency is to perform a tracheotomy early or not at all. In the entire series, only 43 cases were intubated without subsequent tracheotomy, as seen in Table 4.

Table 4.

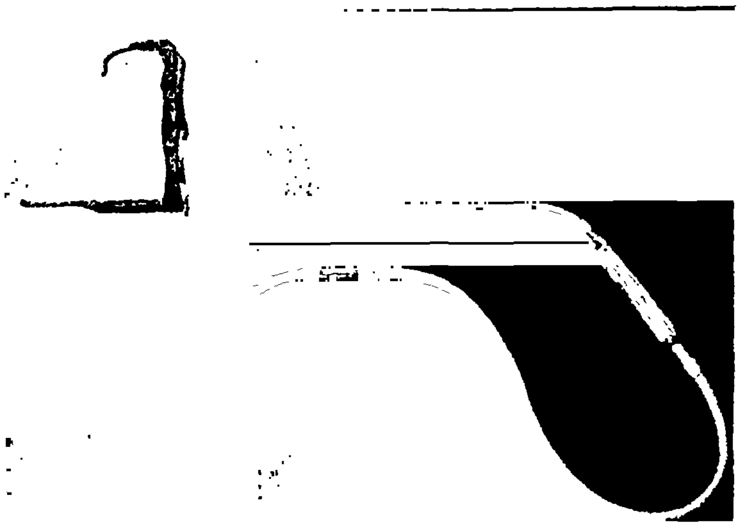


	CROUP	INTUBATION ONLY, NO TRACHEOTOMIES	DEATHS	MORTALITY %
1940	DIPHTHERITIC	3	1	33.3
	NON DIPHTHERITIC	9	2	22.2
1941	DIPHTHERITIC	1	0	0
	NON DIPHTHERITIC	8	1	12.5
1942	DIPHTHERITIC	1	0	0
	NON DIPHTHERITIC	2	0	0
1943	DIPHTHERITIC	2	1	50.0
	NON DIPHTHERITIC	2	0	0
1944	DIPHTHERITIC	1	0	0
	NON DIPHTHERITIC	1	0	0
1945	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	0	0	0
1946	DIPHTHERITIC	3	1	33.3
	NON DIPHTHERITIC	1	0	0
1947	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	5	0	0
1948	DIPHTHERITIC	1	0	0
	NON-DIPHTHERITIC	3	0	0
1949	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	0	0	0
TOTALS	DIPHTHERITIC	12	3	25.0
	NON DIPHTHERITIC	31	3	9.6
	GRAND TOTALS	43	6	13.9

Consistent with this trend away from surgical intervention is the marked decrease in the incidence of laryngoscopies. In 1940, of 155 nondiphtheritic croup admissions, 137 were laryngoscoped and 60 were suctioned; by contrast, in 1948, of 77 similar admissions, only 14 were laryngoscoped and suction was used in only nine of these (see Table 1).

Various authors contributing to this subject have both written and implied that direct laryngoscopy can now be performed quickly and with such ease and gentleness that it should head the list of routine procedures. There can be no doubt that gentle technique and skill are essential to the successful management of these cases; however, good judgment still is of major importance and gives one a "feel" of the case. This "feel" has been developed to a high degree in some of the nursing supervisors who have been in long and intimate contact with a great number of croup patients. It has been invaluable in helping them sense any change in the patient's condition and in alerting the medical personnel so that appropriate measures could be taken as soon as possible before precious time and the patient's resistance have been sacrificed.

It is difficult to imagine a "completely harmless" laryngoscopy. To subject a severely ill, apprehensive child with a temperature of 104° F. or more to forceful restraint by "mummifying" and to hold the head in such a position as to favor struggle and increased pharyngeal and laryngeal congestion can hardly be considered harmless. In acute laryngeal obstruction the instrumentation may precipitate complete respiratory obstruction; therefore, in the acute emergency, direct laryngoscopy should be attempted only if the diagnosis cannot be established otherwise, or if surgical relief of the obstruction through direct examination and aspiration, intubation or tracheotomy is being considered.[3] Suction equipment and a readily available airway are essential before this procedure should be contemplated. The Willard Parker suction tube devised by Tolle is ideal for this purpose as it both aspirates the trachea and is the best possible airway. This lifesaving tube may be left in place to meet the immediate emergency



and is essential as an airway during tracheotomy. The presence of an airway of this type or a small bronchoscope in the trachea during tracheotomy makes the operation an unhurried, tranquil procedure. It is probably the greatest single factor in lowering the mortality rate of tracheotomy *per se* and in avoiding mediastinal emphysema and pneumothorax.

Sufficient inspection of the throat to rule out diphtheria and retropharyngeal abscess can usually be done easily and quickly by means of a well placed light, a head mirror and a tongue depressor. With the child in as comfortable a position as possible, a careful examination for evidence of diminished breath sounds and areas of congestion should be made. All physical signs of obstruction to the air passages such as suprasternal, supraclavicular, epigastric and intercostal retractions should be carefully appraised and a close watch kept over the patient for any change in these classical and informative signs. Increases of the pulse, respiration and temperature are indications of the severity of the infection but are not useful as indications for tracheotomy; however, fatigue and exhaustion are important and must be carefully evaluated. Tracheotomy should not be delayed if exhaustion is increasing.

Unless tracheotomy is contemplated, it is preferable to keep the child as quiet as possible with steam inhalations, oxygen, or both. The question of steam inhalations versus oxygen remains a controversial one and both have their ardent supporters.

Steam is soothing to the mucous membranes. It helps keep the secretions moist and facilitates expectoration; however, when steam is used, care should be taken to keep the temperature of the room quite low since overheating interferes with the child's rest and is generally debilitating. Mechanical steam or vapor has the desired humidity without the disadvantages of the heat.

Asthma may be overlooked in infants. Allergy plays an important part in a considerable number of cases. Among the types seen in this group are spasmodic croup and allergic edema of the hypopharynx, epiglottis and larynx. Steam usually makes these patients worse. They do much better in a cool oxygen tent. To illustrate this point, it is known that many of these cases improve during the cold ambulance ride to the hospital and they arrive in much better condition than when they left home. Epinephrine solution, 1:1,000, should be given hypodermically, 0.25 cc. to an infant of one year and 0.5 cc. to older children. Epinephrine is well tolerated by children. Whisky, which has often been given to croup cases for its sedative effect, is contraindicated in allergy as it produces vasodilation. An antihistaminic elixir is preferable.

The majority of cases of laryngotracheobronchitis are best treated with a combination of cool vapor and oxygen as advocated by Davison.[6] It has been routine for some time at Willard Parker Hospital to administer penicillin, sulfadiazine and streptomycin to each croup case as soon as possible after admission. The usual dose is 50,000 to 75,000 units of aqueous penicillin (for quick absorption into the blood stream) plus 150,000 to 200,000 units of procaine penicillin in oil, the latter to be repeated every 12 hours. An initial injection of 250 mgm. to 375 mgm. of streptomycin is repeated every six hours. The child is also given 1.5 gr. of sulfadiazine per pound of body weight every 24 hours, one-half of the 24-hour dose being given initially and then one-sixth of the daily dose every four hours. The oral preparation used is in a liquid form, containing 0.5 gm. of sulfadiazine and 1.5 gm. of sodium lactate in each 5 cc. Since streptomycin is seldom given for a period of longer than one week, no serious toxic symptoms have been encountered. Aureomycin and chloromycetin are also being used at the present time but the results as yet are not conclusive.

There were 98 tracheotomies in this series, 12 in the diphtheritic group and 86 in the nondiphtheritic. There were two deaths in the former group with a mortality rate of 16.6 per cent, and 10 deaths among the nondiphtheritic cases with a mortality rate of 11.6 per cent. The number of cases, deaths and mortality rate for each year is shown in Table 5. The overall mortality rate of 11.6 per cent for 1940-1949 compares very favorably with the 38.6 per cent mortality rate for the 10-year period, 1930-1939.

Table 5.

	GROUP	TRACHEOTOMY WITH OR WITHOUT INTUBATION AFTER ADMISSION	DEATHS	MORTALITY %
1940	DIPHTHERITIC	4	1	25.0
	NON DIPHTHERITIC	24	5	20.8
1941	DIPHTHERITIC	4	1	25.0
	NON DIPHTHERITIC	13	1	7.7
1942	DIPHTHERITIC	1	0	0
	NON DIPHTHERITIC	12	1	8.3
1943	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	13	2	15.4
1944	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	6	0	0
1945	DIPHTHERITIC	1	0	0
	NON DIPHTHERITIC	7	1	14.2
1946	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	3	0	0
1947	DIPHTHERITIC	2	0	0
	NON DIPHTHERITIC	1	0	0
1948	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	4	0	0
1949	DIPHTHERITIC	0	0	0
	NON DIPHTHERITIC	3	0	0
TOTALS	DIPHTHERITIC	12	2	16.6
	NON DIPHTHERITIC	86	10	11.6
	GRAND TOTALS	98	12	12.2

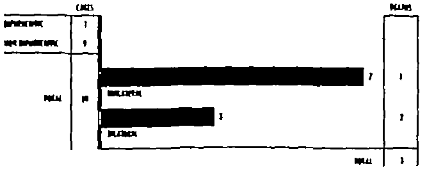
As mentioned above, although there was a slight increase in the frequency of tracheotomy, there was a marked decrease of intubation. In part this may be explained by the tendency to perform the tracheotomy earlier without resorting to intubation and in part to the greater risk and danger from suffocation should the child cough up the intubation tube unexpectedly. With the constant decrease in the number of cases requiring intubation the hospital resident staff is deprived of sufficient opportunity required to attain dependable skill in meeting these emergencies.

In 36 of the patients requiring tracheotomy, the obstruction was due to supraglottic edema, a factor which automatically eliminates the indication for intubation. Slightly more than half of the tracheotomy cases were previously intubated, as shown in Table 4, but at present there is an increasing trend away from intubation.

The incidence of pneumothorax in the total of 98 tracheotomies was 10.4 per cent—nine nondiphtheritic and one diphtheritic (see Table 6). Three were bilateral, two of whom died, and seven were unilateral with one death. Of the unilateral, six were on the left and one on the right side. It is believed that the most important prophylactic measure against pneumothorax is the routine preoperative insertion of an airway, a suction tube, or a small bronchoscope into the trachea. During the operation, then, the child does not struggle against an obstructed larynx, thus drawing in or expelling air from

Table 6.

[illegible]



the chest cavity through the fascial planes of the tracheotomy incision or rupturing the pleura, as a continuous airway is maintained.

The youngest patient in this series was a six weeks old child and the oldest a 53-year-old man. The majority of cases were in the one- to two-year age group. There was a predominance of males (69 per cent) over females (31 per cent), a factor which is quite consistent in most series reported in the literature.

November, December, February and March were the months of greatest incidence.

BACTERIOLOGY.

The bacterial etiology of laryngotracheobronchitis has been a subject of much controversy and will probably remain so for the immediate future. Many authors contend, as Baum does, that the condition is caused by a virus infection, probably influenzal, with a secondary invasion by various pathogens, most frequently the streptococcus.[7] Others contend that most cases of severe supraglottic edema, especially those involving the epiglottis, are caused by the influenza bacillus. Of the cultures taken from the throat, larynx and trachea of our series of 1,077 cases, streptococcus was reported 472 times, staphylococcus 374 times, and an occasional case of influenza bacillus. The influenza bacillus occurred in pure culture in only two cases, both of which were fatal. Their case histories follow:

A three-year-old boy was brought to the "walk-in room" of Willard Parker Hospital by his parents. He was in extreme respiratory distress, and before relief could be given he became cyanotic and expired. Emergency tracheotomy and artificial respiration were immediately carried out, but he failed to respond. The postmortem report in part: "The epiglottis is extremely enlarged and is covered with a purple edematous mucosa; the aryepiglottic folds are also cyanotic and edematous. There is a marked supraglottic edema, the false cords are edematous and congested; the true cords are intact and the ventricles are clear. The subglottic mucosa is only slightly congested and is covered with a minimal amount of mucus. The bronchi contain no mucus. Culture from the larynx was H. influenza and from the blood indicating an H. influenza bacteremia."

The other case was that of a four-year-old white girl with a two-day history of illness. She was brought to the hospital in extreme respiratory distress, marked cyanosis and retractions. She died within five minutes after arrival. An airway tube was passed into the larynx and artificial respiration and epinephrine were given without response. Postmortem examination revealed extreme edema of the epiglottis, aryepiglottic folds and false cords. Cultures from the larynx and also from the blood were positive for B. Pfeiffer.

SUMMARY AND CONCLUSION.

The past decade has seen approximately a 20 per cent decrease in the incidence of these croup cases. The 80 per cent reduction of the overall mortality rate along with the present low mortality of tracheotomies is most gratifying. During this time there has been a marked decrease in the incidence of surgical and mechanical intervention, especially intubation.

There is every reason to anticipate that the future clinical and therapeutic advances will continue toward the ultimate conquest of this vicious disease.

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A THIRD STATISTICAL STUDY OF AUDITORY TESTS IN RELATION TO THE FENESTRATION OPERATION.[*]

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INTRODUCTION.

This report is a continuation of a long-term study[1-6] of the results of tests of hearing conducted on all of the patients on whom the fenestration operation has been performed by one of us (T.E.W.) at McMillan Hospital (Washington University). It deals with over 100 cases (No. 217 – No. 332 inc.), operated upon for the first time between December 2, 1947 and July 15, 1949. (No "revision" operations are included.) Five cases are arbitrarily omitted: three because of lack of full familiarity with English (their mother-tongue was Spanish) and two with psychological difficulties who did not cooperate fully in some of the tests. The mean age of the group was 32.5 years with a standard deviation (σ) of ± 8.9 years and a range from 10 to 64 years.

This study is particularly concerned with interrelationships among the speech tests and pure tone tests that were administered to these patients as part of the routine of pre-operative testing and post-operative follow-up.

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[*] This study was begun under Contract N6onr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under Contract V1001M-577 between Veterans Administration and Central Institute for the Deaf. The routine testing and the collection of data were later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University). The work there is supported by a grant from the Hofheimer Foundation.

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II. PROCEDURE.

The routine of auditory testing was planned to compare·

a) Auditory Test No. 14 (recorded spondaic words, constant level) with Auditory Test No. 9 (recorded spondaic words, diminishing levels),

b) the presentation of spondaic words by "live voice" with the usual (recorded) version of Auditory Test No. 9,

c) "live voice" presentation of the PB word lists with the recorded (Hughes) version of the same lists as a means of determining a patient's maximum articulalation score ("PB-max"), and

d) the relationships between speech tests and the puretone audiogram in relation to the effects of the fenestration operation.

The tests employed have been described in detail elsewhere.[1,2,3,7]

The routine of testing was as follows:

1) *Pure-tone audiometry by air and bone conduction,* performed in the Ear, Nose and Throat Clinic at McMillan Hospital according to the usual clinical routine.

2) Speech audiometry was conducted at Central Institute for the Deaf from December 2, 1947 to September 27, 1948. The sequence of tests during this period was:

  a) Threshold for speech by Auditory Test No. 9 (spondaic words).[7]

  b) Maximum articulation score (PB-max) by recordings of phonetically balanced lists of monosyllables[4] made at Central Institute for the Deaf (voice: R. Hughes). The usual level was 110 db re 0.0002 dyne per cm^2, although for some seriously hard-of-hearing ears 120 db was also used and for others, following fenestration, 100 db sufficed. If more than one level was used the higher score was taken as the "PB-max". The object of the test was to measure the *maximum* articulation score that the subject could attain.

c) Articulation score (PB lists) at a level 85 db above 0.0002 dyne per cm^2 (PB-85).

First the operated, then the unoperated ear was tested at each step.

On September 27, 1948 the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University) was opened. The routine of speech audiometry in the Hofheimer Laboratory from September 27, 1948 to June 29, 1949 was as follows:

a) Threshold by Test No. 9.

b) Threshold for spondees by live voice (Threshold, L. V.) following the vocabulary and routine of Test No. 9.

c) Maximum Articulation Score (recorded lists) at a level at least 35 db above the Threshold by Test No. 9 if such a level was tolerable, otherwise at the highest tolerable level.

d) Maximum Articulation Score by live voice (PB-max, L.V.) using the PB lists and at the same level as *c*.

e) PB-max (recorded) repeated at the same intensity or, occasionally, a little higher.

About half-way through this series the order of *c* and *d* was reversed. The entire testing session lasted about one and one-half hours.

On June 30, 1949 Auditory Test No. 14 (recorded spondaic words at constant level) was substituted for the measurement of threshold by live voice. The routine was continued without further change until the series was closed on April 1, 1950.

The same calculations were made on the differences within each post-operative session. Here there was some slight evidence (by the "t" test) for a poorer performance (by 3.5 per cent) on the second test by the subjects whose PB-max score was below 85 per cent. This questionable downward drift suggests fatigue rather than learning.

The PB-max scores are also reasonably stable from session to session. In the unoperated ear (and omitting all tests made at a level less than 35 db above the threshold for Test No. 9) the average change (for 62 cases with PB-max 85 per cent or higher) is an *improvement* of 1.77 per cent. This is *not* significant by "t" test or sign test. The correlation coefficient *(r)* is + .26. The standard error of measment (σ_{meas}) is 4.54 per cent. (Note that in this series the PB-max is the average of two scores obtained in each session.)

As in the previous series[3] the PB-max scores are less stable when they are below 85 per cent. (The standard error of measurement (σ_{meas}) for the 43 cases with scores below 85 per cent is 6.10 per cent.) The *r* is higher (+ .53) but there is a slight *deterioration* of PB scores from one session to the next by 2.88 per cent (N = 43). This change is on the borderline of significance (5 per cent level) by the sign test and nearly so by the "t" test. The direction of the change is, however, opposite to the slight upward drift found in our previous series. All in all, therefore, *we have no evidence of a significant systematic drift in the PB-max scores from one session to the next.*

3. *Maximum Articulation Score:* "Live voice" (E.E.) vs recorded (R. Hughes).

There is an average difference of 8.11 per cent (N = 90) in PB-max in favor of "live voice" (Eldert) over the recorded test (Hughes). There is no doubt as to the significance of the difference as the difference was in the same direction *in every case.*

IV. GAINS FOLLOWING FENESTRATION.

The gains in decibels following the fenestration operation have been analyzed in full elsewhere,[5] but several correlations among the gains measured in various ways are of some interest.

1. Relation to Size of Fenestra.

Measurements were made at operation of the size of the fenestra produced in the horizontal semicircular canal, and in general the surgeon attempted to make as large an opening as seemed practical. The reason for this was to reduce the likelihood of closure. The incidence of closure has been analyzed elsewhere;[6] but we have also examined our data to determine whether there is any relation between the size of the fenestra and the resulting improvement in the threshold for speech.

Correlations were calculated between the improvement in the threshold for speech and *a)* the width of the opening, *b)* the length of the opening, and *c)* the area of the opening (proportional to the product of length by width). The first was calculated as tri-serial eta ($\eta = .23$) and the two latter as product moments ($r = -.05$ and $r = .08$ respectively). The number of cases (N) was 99 in each case. None of the correlations is statistically significant. This negative result is not surprising as the variations in size were not great, and particularly as we have shown elsewhere that the gain for speech depends primarily on the amount of conductive deafness that the patient happens to have at the time of the operation.

Inasmuch as the most predictable result of the fenestration operation is not the *improvement* in the threshold for speech but the *absolute level* of the (post-operative) threshold[5] it seemed possible that some relation between the immediate post-operative threshold and the size of the fenestra might be found. The calculations were based only on the cases diagnosed as pure conduction deafness in order to avoid contamination by nerve deafness, which is not affected by the operation. Cases in which some "surgical accident" occurred were also omitted. The calculations (tri-serial eta and analysis of variance) showed *no* significant correlation between the postoperative thresholds and either the width, the length or the area of the fenestras as measured by the surgeon. It should be noted, however, that the fenestras were all between 1.0 and 1.5 mm. in width and between 2.5 and 4.0 mm. in length.

2. *Gain for Speech vs Gain by Air Audiogram.*

The correlation between the immediate gain for speech (Test No. 9, measured approximately three months after operation) and the gain for pure tones (average for 512, 1024, 2048 and 2896 cps, measured at the same sessions as the speech thresholds) is $r = +.74$ (N = 103). This is exactly the same coefficient as we found in both our first and our second series.[1,3]

The mean gain by Test No. 9 in the present series is 23.52 db; by air audiogram it is 19.98 db. As before, the gain measured by Test No. 9 is significantly larger (beyond the 1 per cent level of confidence by the "t" test) than when it is measured by averaging the pure-tone audiogram over the speech range. The discrepancy of 3.54 db in favor of the direct measurement is practically identical with the similar discrepancies we found in our two previous series (4.06 and 3.40 db respectively).[1,3]

3. *Gains for Different Frequencies of Pure Tone.*

As before, the gains at 512 and 1024 cps are practically the same as for Test No. 9 while the gains for 2048 and 2896 cps are significantly less.

The mean gains at the various frequencies (N = 106) are:

512 cps = 22.45 db
1024 cps = 23.25 db
2048 cps = 20.24 db
2896 cps = 11.60 db

The only differences that are not significant are those between 512 and 1024 and between 512 and 2048 cps. This finding confirms our previous results.

It seemed possible that the smaller gains at 2048 and 2896 cps might be only a temporary deficit. We therefore compared the average gains for the air audiograms as a whole and for 2048 cps separately as measured 6 to 15 months after operation. The gains were not significantly greater than the immediate gains of the same group of patients (N = 40). Neither did the gains measured 12 months or more after operation differ significantly from the results of the "immediate" tests (N = 12, sign test). Likewise the relation between the gain for speech (Test No. 9) and the gain by average air audiogram showed *no* significant difference after 11 months from the relations calculated from the immediate gains and given in sub-section 2 above. (N = 66; "t" and sign tests). We conclude that the gains measured within the first three months remain substantially stable on the average during the following year.

We still have no explanation to offer for the relatively smaller gains at the higher frequencies following fenestration.

4. Prediction of Gain for Speech.

We have shown in a previous paper[5] that the gain for speech following fenestration depends primarily, as we should expect, on the amount of conductive loss present before the operation. To test this conclusion further we have calculated the correlation between gain for speech (Test No. 9) and the pre-operative conductive loss. The measure of conductive loss was the pre-operative difference between the loss by air conduction and the loss by bone conduction (average of 512, 1024, 2048 and 2896 cps). The value of r (N = 50) is + .45. This is a statistically significant correlation and confirms the concept elaborated in our previous paper.

5. *The Effect of Fenestration on Maximum Articulation Score.*

The fenestration operation did not significantly alter the maximum articulation scores of the operated ears (N = 108). The observed differences were *not* significantly different by either "t" test or sign test, not even for the sub-group (N = 23) whose pre-operative tests were performed at levels slightly less than 35 db above their No. 9 thresholds. This constancy of the maximum articulation score was expected theoretically and agrees with our previous results.[3]

Incidentally, the average PB-max (for cases tested 35 db or more above the threshold for Test No. 9) of the *operated* ears was compared with the corresponding average PB-max of the *unoperated* ears. The difference of 1.75 per cent in favor of the latter is *not* significant.

DISCUSSION.

This statistical study of the auditory tests administered to approximately 100 additional patients on whom the fenestration operation has been performed has yielded little that is new but it confirms quantitatively as well as qualitatively many of the conclusions derived from our previous studies. In no case where the same points were re-examined has it been necessary to modify significantly our earlier conclusions. In view of this stability of the results it should not be necessary to repeat these particular studies until some significant change in technique is introduced, either in the operation itself or in the tests of auditory function.

In addition to the data here reported we have also analyzed with some care the articulation scores obtained with our recorded versions of the PB lists. The results of this study will be reported in the near future.

SUMMARY.

Speech tests and pure tone tests of hearing were administered to over 100 nearly consecutive patients before and after

the fenestration operation. The reliability, the standard error of measurement, and other statistical indices were calculated. The results agree very closely with those of our previous similar studies.

The spondaic word tests are highly reliable as tests for the threshold for speech. The two recorded versions, Test No. 9 (descending levels) and No. 14 (constant level), and administration by "live voice" are all practically interchangeable.

The maximum articulation score (PB lists given at least 35 db above the threshold for Test No. 9) is significantly higher when the lists are given by "live voice".

The maximum articulation scores ("PB-max") show no evidence of a learning factor from one session to the next under the conditions of our clinical routine testing.

The maximum articulation score is slightly but significantly lower for the patients with some high-tone nerve loss.

The articulation score at the arbitrary level of loud speech, i.e. 85 db re 0.0002 microbar, is a less stable measure than the maximum articulation score.

No significant correlation was found between the size of the fenestra (measured at operation) and either the threshold for speech or the gain for speech following the operation.

As in our previous studies the average gain from fenestration is less for frequencies 2048 and 2896 cps than for frequencies 512 and 1024. The gains at the lower frequencies agree very closely with the gain measured by Auditory Test No. 9. The relative deficit at the higher frequencies is still present after one year and is probably permanent.

The fenestration operation does not significantly alter the maximum articulation score of either ear.

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HEADACHE AND VERTIGO ASSOCIATED WITH HYPOGLYCEMIC TENDENCY.*†

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Considerable detailed information regarding the various aspects of spontaneous hypoglycemia is contained in the medical literature since Harris,[1] in 1924, described a fairly characteristic group of symptoms experienced by individuals with abnormally low blood sugar levels. Little or no attention has been placed on an even larger group of patients whose symptoms are directly related to lowered blood sugar levels. Headache and, to a lesser extent, vertigo are common symptoms experienced by this latter group. In practically every case the headache and vertigo will be reproduced during the course of the five-hour glucose tolerance test.

The etiologic classification of spontaneous hypoglycemia as set forth by Conn[2] applies to this group of patients. The available information placed these patients in the functional or organic hepatogenic groups.

ETIOLOGIC CLASSIFICATION OF SPONTANEOUS HYPOGLYCEMIA.

I. Organic—recognizable anatomic lesion.
  A. Hyperinsulinism.
    1. Pancreatic island cell adenoma.
      a. Single.
      b. Multiple.
      c. Aberrant.

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2. Pancreatic island cell carcinoma.
    a. Localized.
    b. With metastases.

3. Generalized hypertrophy and hyperplasia of the islands of Langerhans.

B. Hepatic disease.

1. Ascending infectious cholangiolitis.
2. Toxic hepatitis.
3. Diffuse carcinomatosis.
4. Fatty degeneration or "fatty metamorphosis."
5. Glycogenosis (von Gierke's disease).

C. Pituitary hypofunction (anterior lobe).

1. *Destructive lesions (chromophobe tumors, cysts).*
2. Atrophy and degeneration (Simmond's disease).
3. Thyroid hypofunction (? secondary to pituitary hypofunction).

D. Adrenal hypofunction (cortex).

1. Idiopathic cortical atrophy.
2. Destructive infectious granulomas.
3. Destructive neoplasms.

E. Central nervous system lesions (Hypothalamus or brain stem interference with nervous control of blood sugar).

II. Functional—no recognized anatomic lesion but explainable on basis of unusual somatic function.

A. Hyperinsulinism (imbalance of the autonomic nervous system); hypoglycemic fatigue; nervous hypoglycemia; functional hypoglycemia; reactive hypoglycemia.

- *B*. Alimentary hyperinsulinism (rapid intestinal absorption).
    - *1*. After gastroenterostomy.
    - *2*. After gastric resection (partial or total).
- *C*. Renal glycosuria (severe degrees of low renal threshold for dextrose).
- *D*. Lactation.
- *E*. Severe continuous muscular work.

III. Miscellaneous.

- *A*. Factitious (surreptitious insulin administration).
- *B*. Postoperative hypoglycemia.
- *C*. Severe inanition.
- *D*. Unknown.

In order to associate some relationship between the patient's symptoms and the blood sugar levels, the standard preparatory diet was not used, and the patient was instructed to remain on his usual diet until after the glucose tolerance test was made.

The nature of the diet preceding the test has a marked influence on the results of the test in both animal and man. The restriction of carbohydrates in man results in a marked decrease in ability to utilize carbohydrates and produces an exaggerated glucose tolerance curve. A diet high in carbohydrates, low in protein, produces an increased tolerance and the curves are low and flat. A diet high in fats produces a much greater and more prolonged hyperglycemia when glucose is given than does one high in carbohydrates. The ingestion of large amounts of protein is followed by very little rise in the blood sugar. The long period required for the absorption and conversion of protein gives a steady supply of dextrose over a fairly long period of time, resulting in very little elevation and secondary fall of blood sugar levels. These facts account for the therapeutic effectiveness of a high protein diet in cases of functional hypoglycemia.

The effect of high and low carbohydrate diets preceding the oral administration of glucose is shown in Fig. 1.[3]

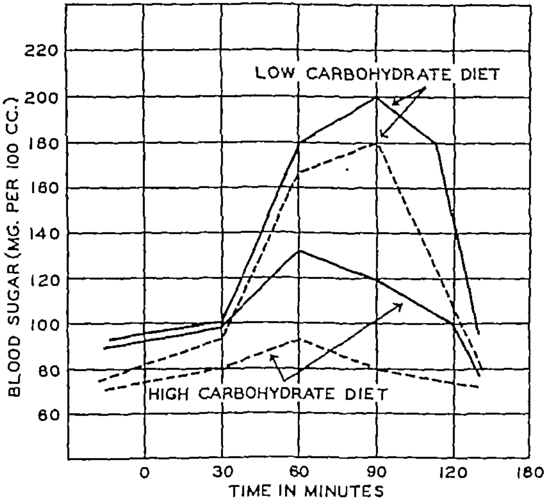

Fig. 1. The influence of high and low carbohydrate diets preceding the oral administration of 50 gm. of glucose ————, capillary blood sugar, - - - - -, venous blood sugar. (Courtesy, Kolmer: Clinical Diagnosis by Laboratory Examinations, 2nd ed. New York, N. Y.: Appleton-Century-Crofts, Inc.)

The case histories of 72 patients complaining of headache or vertigo and whose symptoms were relieved by high protein diet were reviewed. In all patients the symptoms were reproduced or aggravated during the five-hour glucose tolerance test.

Forty-five (62.5 per cent) were females. Thirty (41.8 per cent) of the patients were in the third decade (see Fig. 2). Headache was the chief complaint of 70; vertigo, in two. Both vertigo and headache were present in 32 (44 per cent).

The headache was described as being dull in 65 of 65 cases and throbbing in 56 of 59. It was relieved by pressure in 18

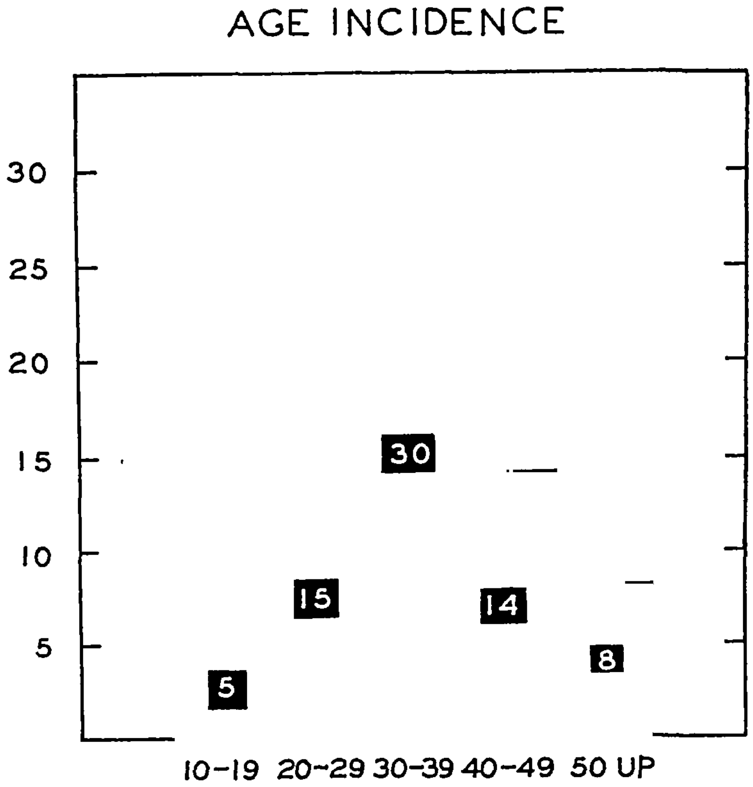

Fig. 2. Age incidence.

of 32 cases. Forty-two of 56 patients were awakened at night by headache, and in 44 patients, 39 had headache before breakfast. In 33 of 48 patients the headache was definitely related to meals.

The headache experienced is visceral in character and is described as being dull, throbbing and is often relieved by pressure. This is the same type of pain that was produced experimentally by Ray and Wolff[1] by mechanical distention of the superficial temporal artery. The same type of pain is experienced when a hypodermic needle is pushed through the wall of an artery or vein.

The glucose tolerance curve, in most instances, was low, especially from the second through the fifth hour. In only 15 cases did the fifth hour level reach the fasting level, and in seven of these 15 the curve was flat. Symptoms were usually produced when the level dropped to 70 or less. In others, the rapidity of the fall of the sugar levels appeared to be the important factor.

The following abbreviated case histories are typical

Case 1 Mrs D B H., age 32 years, was first seen on Aug. 21, 1947, with the chief complaint of vertigo of three months' duration, which was associated with some nausea and seemed to be more or less continuous in character and increased in intensity at times. The patient stated that she was subject to motion sickness Vertigo, nausea and jitteriness were present if a meal was missed. There was no history of headache, but occasionally a sensation of pressure was present in the vertex.

The past medical history was noncontributory The patient's mother had asthma for a number of years.

The ear, nose and throat examination did not reveal any gross deviation from the normal A medical and ophthalmological examination revealed nothing significant

The five-hour glucose tolerance test gave the following results Fasting—80 mg per cent, one-half hour—136 mg per cent, one hour—104 mg per cent, two hours—73 mg. per cent, three hours—70 mg. per cent, four hours—58 mg per cent, five hours—59 mg. per cent. During the glucose tolerance test, the patient experienced a considerable amount of vertigo associated with headache.

The patient was given a high protein low carbohydrate diet and was instructed to eat between meals and before bed time. Within a very short time, all symptoms had disappeared and she had remained free from attacks when last seen on Jan 5, 1949, unless she had changed her diet.

Case 2 Mrs. E. F D, age 35 years, was first seen on Nov 8 1948, complaining of occipital and left temporal headache which was dull and throbbing in character and not relieved by pressure The patient's symptoms had been present for four years but had become more severe for the last years. She was occasionally awakened at night with headache, and it was usually present in the morning before breakfast Occasional vertigo was associated with the headaches The patient was quiet fond of soft drinks and consumed a number each day

The past medical history was noncontributory except for symptoms suggestive of hay fever

The ear, nose and throat examination was essentially negative except for some slight polypoid changes on the posterior tips of the inferior turbinates and the presence of a small polyp which was removed from the left middle meatus

Medical examination failed to reveal abnormalities which could be associated with her headaches

The five-hour glucose tolerance test revealed the following: Fasting—89 mg. per cent; one-half hour—115 mg. per cent; one hour—102 mg. per cent; two hours—103 mg. per cent; three hours—91 mg. per cent; four hours—68 mg. per cent; five hours—68 mg. per cent. During the glucose tolerance test, the patient experienced headache similar in character to that which she had been having for the past four years.

A diet high in protein and low in carbohydrates was prescribed and her symptoms gradually disappeared and when last seen on May 2, 1949, she was symptom free.

Case 3: Mrs. M. E. G., age 37 years, was first seen on Jan. 7, 1949, complaining of dull, throbbing pain in and about the left cheek and eye for approximately one year. The patient was overweight and had been on a strict diet for some time.

A medical examination and neurological consultation revealed nothing eventful.

The five-hour glucose tolerance test revealed the following: Fasting—103 mg. per cent; one-half hour—198 mg. per cent; one hour—222 mg. per cent; two hours—193 mg. per cent; three hours—140 mg. per cent; four hours—69 mg. per cent; five hours—65 mg. per cent. During the fourth and fifth hours of the glucose tolerance test, the patient experienced pain similar to that which she had had for the past year.

The patient was placed on a high protein and low carbohydrate diet. Her symptoms quickly disappeared and when last seen on May 3, 1949, she was completely free of her symptoms.

Case 4: Miss P. A. H., age 31 years, was first seen on July 4, 1949, complaining of frontal headache which was dull, throbbing in character and was relieved by pressure. The patient was often awakened with headache between 12:00 and 2:00 o'clock A. M. The patient experienced a similar type headache when a meal was missed and some jitteriness was associated with the headache. The patient had been on a diet low in protein.

The ear, nose and throat examination was essentially negative and medical examination was noncontributory.

The five-hour glucose tolerance test revealed the following: Fasting—66 mg. per cent; one-half hour—98 mg. per cent; one hour—125 mg. per cent; two hours—89 mg. per cent; three hours—67 mg. per cent; four hours—41 mg. per cent; five hours—64 mg. per cent. During the five-hour glucose tolerance test, the patient experienced similar headache and was quite nervous and felt faint throughout the duration of the test.

She was placed on a high protein, low carbohydrate diet with between meal feedings. Marked improvement was noted in a short time and when last seen in November, 1950, she was having occasional difficulty.

SUMMARY.

1. Headache and vertigo are common symptoms associated with lowered blood sugar levels.

2. The headache is described as being dull and throbbing in character. It is often present at night and may be related to meals.

3. Patients in the functional group respond well to a diet high in protein and low in carbohydrates. Supplementary feedings may be necessary.

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MORE COMPLETE ADENOIDECTOMY TO OVERCOME INTERFERENCE WITH NASAL BREATHING BY THE SALPINGONASAL AND SALPINGOPALATINE FOLDS.*

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Most experienced otolaryngologists consider tonsillectomy in children as a relatively simple procedure, but the adenoidectomy and concluding hemostasis as posing a more difficult problem. During the past 10 years, one of us (E. R.) has been using the modified Coakley[1] slip knot tie to control the bleeding. At first only the bleeding vessels in the lower part of the wound were tied; but after skill had been acquired, the source of bleeding higher up was investigated.[2]

In the course of searching for these vessels, it was noted that sometimes, even after what was considered a complete adenoidectomy with LaForce adenotome and curette, there remained, anterior to the pharyngeal tonsil, a thick rugged mass which could not be removed with these instruments without endangering the tube.

The pharyngeal tonsil is defined by Shambaugh in Jackson's book[3] as being "a prominence on the roof and posterior wall of the nasopharynx produced by lymphoid tissue," but this previously mentioned rugged fold was not connected with the pharyngeal tonsil, although it had the appearance of adenoid tissue. It was not located on the posterior wall of the pharynx but was primarily on the palate. The fold was anterior to a catheter inserted into the ostium of the Eustachian tube. The

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finding of the plica salpingopharyngea directly behind the inserted catheter made it clear that this rugged fold was the plica salpingopalatina.

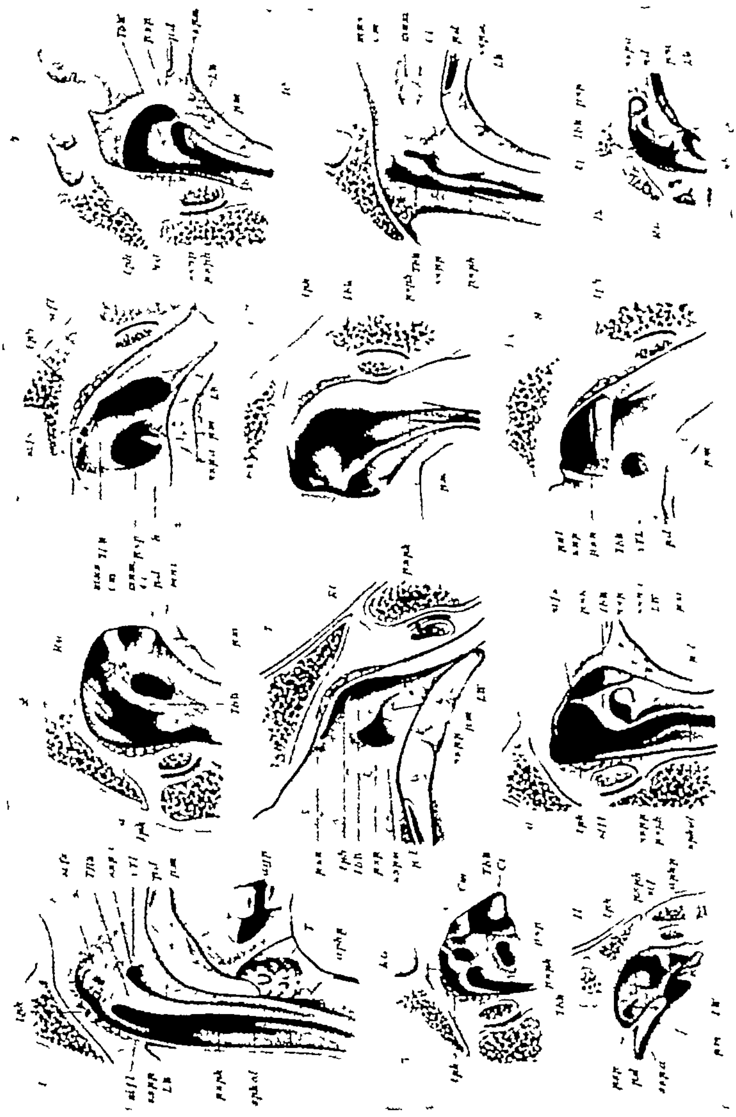
Because of its location, the plica salpingopalatina is not obvious. In most cases it can be seen only during anesthesia in the course of the operation. The best time to look for it is after tonsillectomy but before adenoidectomy. After elevating the soft palate and depressing the main mass of adenoid tissue in the dorsal direction, one can see a fold protruding more or less prominently out of the dorsal surface of the soft palate, quite separate from the adenoid. In most cases this protruding tissue is only 1 or 2 mm. wide. But in 10 of our cases it was at least 9 mm. wide, obstructing one or both choanae and interfering with nasal breathing.

ANATOMICAL CONSIDERATIONS.

Excellent descriptions of the epipharynx are found in Luschka's[4] monograph and in Barnhill's[5] and Testut's[6] anatomies. According to Escat[7] and St. Clair Thomson'[8] lifesize drawings, the height and width of a choana of a newborn averages 6.5 mm. The width in a five-year-old child is 9 mm. and in an adult 13 mm. This means that in a five-year-old, a salpingonasal or salpingopalatine fold wider than 6 mm. is a serious, and one wider than 9 mm. is an absolute obstacle. With hypertrophy of the plicae salpingopalatina and salpingonasalis, the obstruction to nasal breathing will depend upon the location and the extent of the enlargement of the fold as well as the size of the choanae.

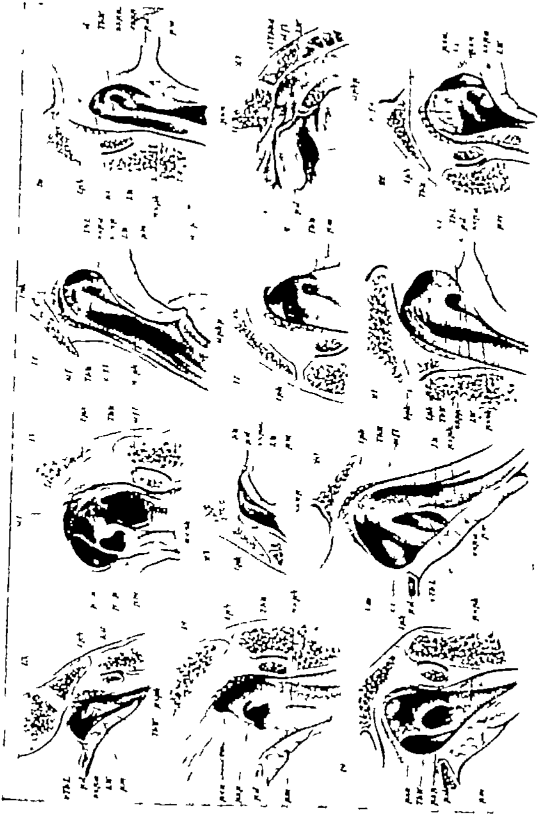
The following lines are excerpts translated from a very accurate anatomical description of the rhinopharynx by Kostanecki:[9]

"The appearance of the anterior lip of the tubal ostium varies greatly in the different specimens (see reproduction. In Figs. 1, 2 and 8 it is small; in Figs. 3, 4, 5, 6, 7, 14, 15, 16 and 17 it is larger.). These variations are caused by developmental differences in strength of a ligament which can be



(a.g.p) arcus palatoglossus; (a.ph.p.) arcus pharyngopalatinus; (b.ph.) bursa pharyngea; (m n.i) meatus nasium inferior; (m.n.m.) meatus nasium medius; (m n s.) meatus nasium superior; (c.i.) concha inferior; (c.m.) concha medium; (c.s.) concha superior; (p.d) palatum durum; (p m.) palatum molle; (p s n) plica salpingonasalis; (p s.p.) plica salpingopalatina, (p n l.) plica nasalis lateralis; (s.ph.) plica

found to be a continuation of the membranous tube into the soft palate. This ligament was named by Tourtual,[10] the anterior salpingopalatine ligament. The shape of this liga-



salpingopharyngea, (R.G.) fossa of Rosenmüller, (si.f.l.) sinus faucium lateralis, (si.f.s.) sinus faucium superior, (si.sph.) sinus sphenoidalis, (si.n.p.) sinus nasalis posterior, (si.ph.o.l.) sinus pharyngo-oralis lateralis, (s.s.p.a.) sulcus palatinus anterior, (s.s.p.p.) sulcus palatinus post., (T) tonsil, (Tb.W.) Torus tubarius, (t.ph.) tonsilla pharyngea, (V.T.L.) anterior lip of the tube, (L.W.) bulge of the levator

ment varies very much in different anatomical specimens. Sometimes it is composed of well developed, tendon-like strands; at other times it is only a fine fibrous continuation

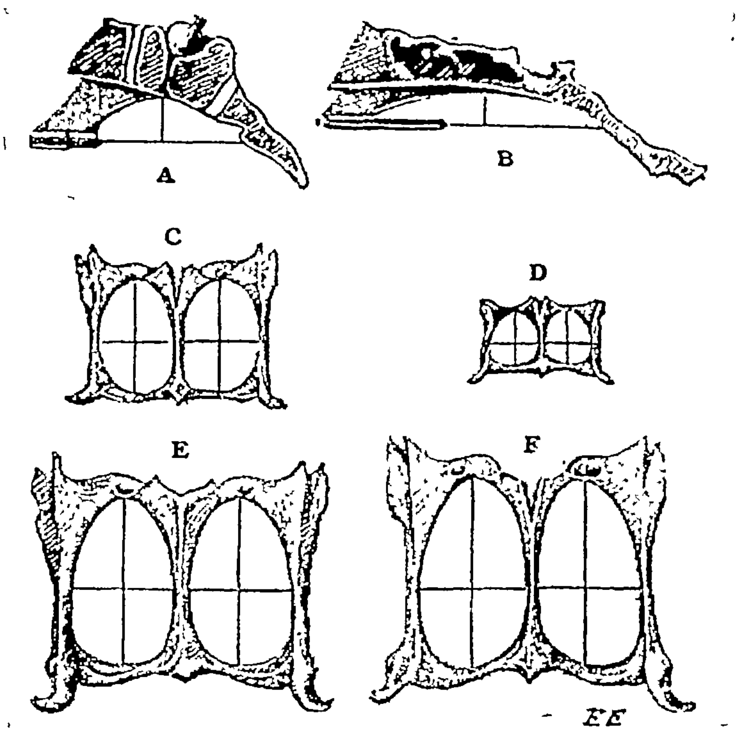


Fig 176 Lifesize drawings of the nasopharynx. From Sir St. Clair Thomson's Diseases of the Nose and Throat (3rd ed., 1926), p. 333. (A) Sagittal section of the nasopharynx at birth, showing persistence of the canal lodging Rathke's pouch (cf. Fig. 2, p 2); (B) sagittal section of the nasopharynx of a dog, (C) choanae of a child aged five; (D) choanae at birth; (E) choanae of an adult female; (F) choanae of an adult male. (Escat.)

of the membranous tube, originating from the lateral part of the tubal cartilage. Sometimes it extends to the midline of the hard palate or it may turn dorsally into the substance of the soft palate, where its fibres blend with the tendon of the tensor veli, the posterior salpingopalatine ligament, and even with the salpingopharyngeal ligament. The result is that it curves about the tubal ostium like an arc.

"The mucosa of the anterior lip of the tube is very stiffly connected with these fibrous bands. Tourtual[10] found that the anterior lip of the tubal ostium is always accentuated by a strong fold of mucosa which he named the salpingopalatine fold. The appearance of this fold depends upon the developmental differences in the strength of the underlying ligament and the thickness of the mucosa covering it. It may be thin and transparent, if the layers of glands and lymph follicles are thin; or it may be thick and very prominent. In its downward course, the salpingopalatine fold runs either directly to the midline of the palate or it describes an arc, the concavity of which faces the ostium.

"Besides this fold which is directed downward, there is another very important fold of the mucosa which originates on the upper part of the tubal torus and is directed upward. This fold was named by Hoffman, the 'salpingonasal fold.' Its thickness is variable. Under the mucosa of the salpingonasal fold he found a continuation of the basilar fibrocartilage composed of small but strong fibres which are strongly blended with the perichondrium. Often the fibres of the anterior palatine ligament unite with these fibres.

"For this reason, the salpingonasal and salpingopalatine folds are often found forming one continuous fold instead of two separate ones (see reproduction Figs. 6, 7, 8, 15, 18 and 19).

"Anterior to the salpingonasal fold, another fold, called by Tourtual the lateral nasal fold, was often found. This continues downward anteriorly to the plica salpingopalatina, so that one sees what appears to be a double salpingopalatine fold (see reproduction Fig. 8). The anterior lip of the tube and the folds originating from it greatly influence the size and form of the choanae."

In our experience, the salpingopalatine fold was always visible. In most cases it originated from the lateral lamina of the tubal torus and formed an arc with the concavity facing the tube. It ended mostly on the dorsal surface of the palatopharyngeal fold. Often the fold did not start at the tube but seemed to originate from the fornix; that is, the salpingonasal and salpingopalatine folds united to make one continuous arc.

PATHOLOGICAL SIGNIFICANCE OF THE HYPERTROPHY OF THE FOLDS.

The importance of the nasopharynx is well emphasized in the excellent symposiums of LeJeune[11] and Dudley.[12] Hollender[13,14] reports the nasopharyngeal findings in a group of 131, and then 140, autopsies. Lyons,[15] Baker,[16] Giddal,[17] Bordley[18] and Dudley[19] write imposing articles confirming the vital importance of the nasopharynx as a source of focal infection, deafness and mouth breathing.

In a brilliant article, Meltzer[20] expresses the opinion that many an irradiation will not be necessary if the lymphoid tissue is removed completely from the nasopharynx.

Our material comprised 155 children ranging from two to 10 years in age. All operations were deferred until at least two weeks after the last acute infection. The cases were not selected at random; many of them were referred by doctors who knew that one of us was trying to resolve these obstructions to nasal breathing by operation.

Thirty-eight cases showed marked hypertrophy of the salpingonasal and salpingopalatine folds. In several cases, the plicae were 10 mm. wide; in one case, 12 mm.; and in one striking instance, 15 mm. wide in its upper part and 10 mm. wide in its lower part.

This last case (No. 103) was that of an eight and one-half year old child whose tonsils and adenoids had been removed six years before. As far as the mother remembered, the child had been a mouth breather all his life. The operation, although performed by a very reliable surgeon, did not relieve the obstruction to the nasal breathing. The child continued to sleep restlessly with an open mouth. There were also several attacks of otitis media, one of them necessitating a paracentesis, last year.

During the office examination, even without anesthesia, two tremendous folds could be seen jutting out from behind the elevated soft palate. These folds tended to meet in the midline during gagging. The diagnosis was obstruction of the nasal breathing caused by hypertrophy of the salpingopalatine folds.

At operation, only a small amount of lymphoid tissue was found on the posterior pharyngeal wall, in the region of the previous adenoidectomy, but the salpingopalatine and salpingonasal folds were huge. They seemed to start well above the tubal ostium and continued onto the soft palate in a curved line. They ended on the dorsal surface of the palatopharyngeal folds. The weight of the removed plicae was 1 gm. The weight of the residual adenoid tissue found on the posterior pharyngeal wall was only 0.5 gm.

Following this operation, the child was able to breathe through his nose without any difficulty.

In seven of the cases the folds were less than 5 mm. wide but so thickened that it was deemed advisable to remove these masses to prevent any possibility of focal infection.

The interference was not always caused by the enlargement of the salpingopalatine or salpingonasal folds. In four of our cases, the salpingopharyngeal fold caused similar symptoms. In the first case (No. 39), the tonsils and adenoids had been very satisfactorily removed three years previously. The child was six years old when seen in consultation. According to his mother, neither before nor after adenoidectomy could the child breathe through his nose.

Office examination revealed a fold visible on each side of the pharynx; this was accentuated by spontaneous elevation of the palate while the tongue was being depressed.

During the operation, these folds were seen to originate behind the tubal ostium and to continue downward onto the posterior pharyngeal wall. Thus they were the hypertrophied salpingopharyngeal folds. Above they were 12 mm. wide and at the level of the base of the uvula, 7 mm. wide. They were removed by tonsil punch. The removed tissue weighed 1.5 gm. The salpingopalatine fold was clearly visible anterior to the above described plica. It was relatively small and was not removed.

After operation the child had no more difficulty in nasal respiration.

In most cases it was found difficult to separate the salpingopharyngeal fold from the pharyngeal tonsil during operation. The adenotome or curette catches it together with the adenoids and removes that part of the fold which is on the posterior wall.

In the second and third of these cases of hypertrophied salpingopharyngeal folds, these plicae, although intimately in contact with the mass of adenoid tissue, somehow evaded the adenotome. In both cases the fold started on the torus but ended on the lateral wall of the nasopharynx. It frequently ends on the posterior wall. For this reason it could not be removed with the LaForce adenotome. In one of these two cases (No. 65), the fold was 10 mm. above and 6 mm. in the lower part.

The third case (No. 177) showed the greatest hypertrophy in our series. It was 18 mm. in its upper part and 10 mm. in its lower part.

INDICATIONS FOR COMPLETE ADENOIDECTOMY.

There are three indications for the removal of these folds: the first is to eliminate any obstruction to nasal breathing. Whether the folds form obstruction or not depends upon the size of the choanae. In young children the choanae are narrow; therefore, even a moderate enlargement of the salpingopalatine or salpingonasal folds could obstruct them. Inspection of the choanae by retronasal mirror is of assistance in the diagnosis of such obstruction, and can be performed if, as sometimes happens, the bleeding stops promptly. In Case 93 the salpingopalatine fold was only 5 mm. wide; nevertheless, the mirror examination revealed that it completely covered one of the choanae.

Since then, as a preliminary working rule, all salpingopalatine or salpingonasal folds of 5 mm. or more in width have been removed.

The second indication for removal is that a large mass of lymphoid tissue in the air passages is an excellent nidus for focal infection. Many of the bacteria carried into the nose by the air are deposited in the film of mucus covering the nasal mucous membrane. This film is moved in the pharynx by the nasal ciliated epithelium. The folds which must be passed constitute a barrier, for often they are covered not by ciliated but by squamous epithelium which does not participate actively in moving the germ-laden film onward into the nasopharynx; therefore, an attempt was made to remove every sizable mass of lymphoid tissue, even though it was not obstructive.

The third indication for removal is that in many cases the folds covered the opening of the Eustachian tube and interfered with the necessary equalization of the air pressure between the tympanic cavity and the pharynx, thus leading toward progressive deafness. They also predisposed toward purulent otitis media, because even in cases of mild infection they prevented easy elimination of the contents of the middle ear through the tubal ostium.

TECHNIQUE OF THE COMPLETE ADENOIDECTOMY.

The basic problem in complete adenoidectomy is to remove all macroscopically visible lymphoid tissue from the nasopharynx *under visual control.* The first step is the removal of the tonsils. This gives the operator more space. Before touching the adenoids, the soft palate is elevated. Any retractor is satisfactory, but the modified Love retractor is preferred because it is light and keeps the hand out of the illuminated territory.

We can see the mass of adenoids filling the space between the posterior wall of the pharynx and the soft palate, but we cannot distinguish the salpingopalatine or salpingopharyngeal folds in this big mass of lymphoid tissue, except when the folds are very bulky.

By pushing the adenoid mass backwards with a curved instrument we can find the salpingopalatine fold easily, even though it is small. A metal curved Eustachian catheter is very useful for this manipulation. If we so separate the soft palate from the adenoid mass, we can observe the salpingopalatine fold on the dorsal surface of the elevated palate as a thin or thick, rugged, uneven fold. If it is large, it is sometimes flattened medially, covering the anterior surface of the pharyngeal tonsil. Sometimes it is flattened laterally, covering the tubal opening.

The next step is the removal of the adenoid mass by the LaForce adenotome. It should be sharp so that one can cut, not tear, out the lymphoid tissue. Tearing results in an uneven, ragged wound, making hemostasis more difficult.

The soft palate is elevated and we usually find that a part of the adenoids, especially on the sides, has not been removed. This can be taken out with LaForce adenotome or with a sharp tonsil punch.

Using suction to clear away the blood, we make careful observations and measure the size of the folds. If the bleeding stops quickly, we use a mirror to examine the relative size of the folds and the choanae.

The next step is of utmost importance. The tubal opening must be located. Not once could it be recognized with the unaided eye, for it is hidden among the abundant lymphoid tissue on the lateral wall. The Eustachian catheter solves this problem. With it inserted into the tubal ostium, the important landmarks can be located easily.

Anterior to the catheter lies the salpingopalatine fold; above, the salpingonasal fold; and behind, the salpingopharyngeal fold and the Rosenmüller fossa. All folds originate from the tube, but the salpingopalatine folds end on the soft palate. The salpingopharyngeal folds end on the dorsal or lateral pharyngeal wall. The usual findings are the salpingonasal and salpingopalatine folds forming a continuous arc, beginning at the fornix and continuing downward on the soft palate, to end most frequently on the dorsal surface of the palatopharyngeal fold. The salpingopharyngeal fold is usually not visible. It is located on the posterior pharyngeal wall and falls into the mouth of the LaForce adenotome together with the adenoids. The lateral pharyngeal wall is often covered with numerous small nodules situated between the salpingopalatine and salpingopharyngeal folds (Barnhill[8]).

Having located the three folds, we remove them if they are too bulky. The most satisfactory instrument seems to be a very sharp conchotome or tonsil punch (No. 2). This instrument can be manipulated with one hand; the other hand is free to lift the palate or to use suction to remove the blood.

This is a great advantage when operating in a deep cavity like the nasopharynx. It eliminates an assistant who has to handle instruments blindly, because the head of the surgeon is blocking the view of the surgical field.

The tonsil punch is used not only for cutting but also for prolongation of the touch sense of the fingers. With it one can feel the hard cartilage below the mucosa; so that the lymphoid tissue can be removed without injuring the cartilage below the salpingonasal fold or the Rosenmüller fossa. Once it has been ascertained by visual examination that everything necessary has been removed, we release the soft palate to its former position.

At this stage of the operation we do not put gauze packings into the nasopharynx. A packing would lift the soft palate and thus elongate the tensor and levator veli palati and put its fibres on tension. The veins which cross these fibres are thus compressed, damming up the blood in the venous capillaries, which cannot collapse, and this in turn tends to prolong bleeding.

HISTOPATHOLOGIC FINDINGS.

All plica tissue removed was sent to the laboratory where it was described, weighed and where paraffin sections were made of representative portions. In the early stages, portions of adenoid tissue were similarly treated. All three folds present in the main, the same basic elements, *i.e.*, lymphoid tissue and glands covered by respiratory epithelium which often shows squamous metaplasia.

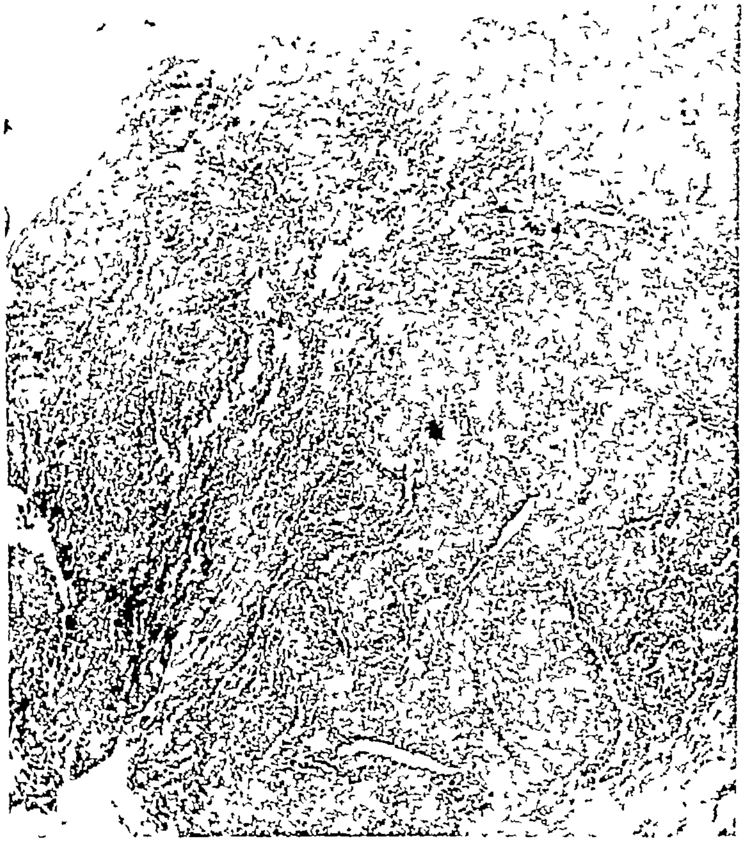


Fig 1 Plica salpingopharyngea from radical adenectomy Part of the plica salpingopharyngea It demonstrates fibrosis, reticulum hyperplasia and the infiltration of squamous epithelium by leucocytes

Histologically, the folds are quite similar to the pharyngeal tonsil at its inferior border. Usually, the major portion of the plica consists of lymphoid aggregates occupying the lamina propria. Subjacent to this are variable numbers of seromucinous glands, separated from the lymphoid tissue by a thin fibroelastic membrane. There is variation in the surface epithelium: some of the specimens bearing squamous epithelium, others being covered by ciliated columnar cells. Epithelial

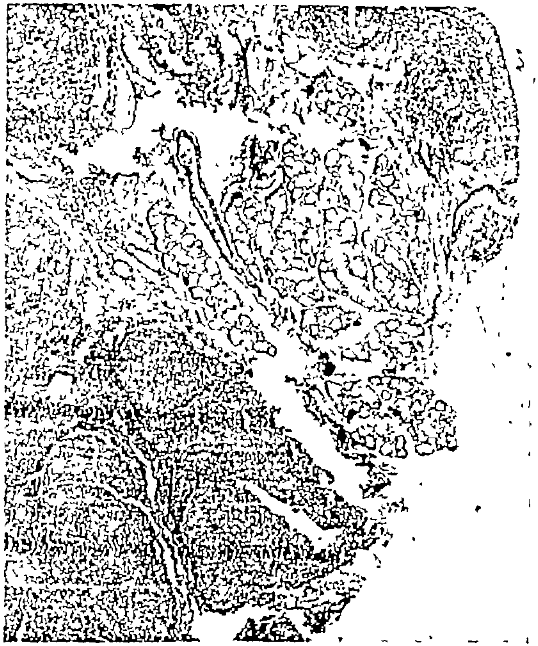


Fig 2 Plica salpingopalatina from radical adenectomy. Plica salpingopalatina presenting glands and lymphoid tissue and predominantly respiratory epithelium with inflammatory infiltration.

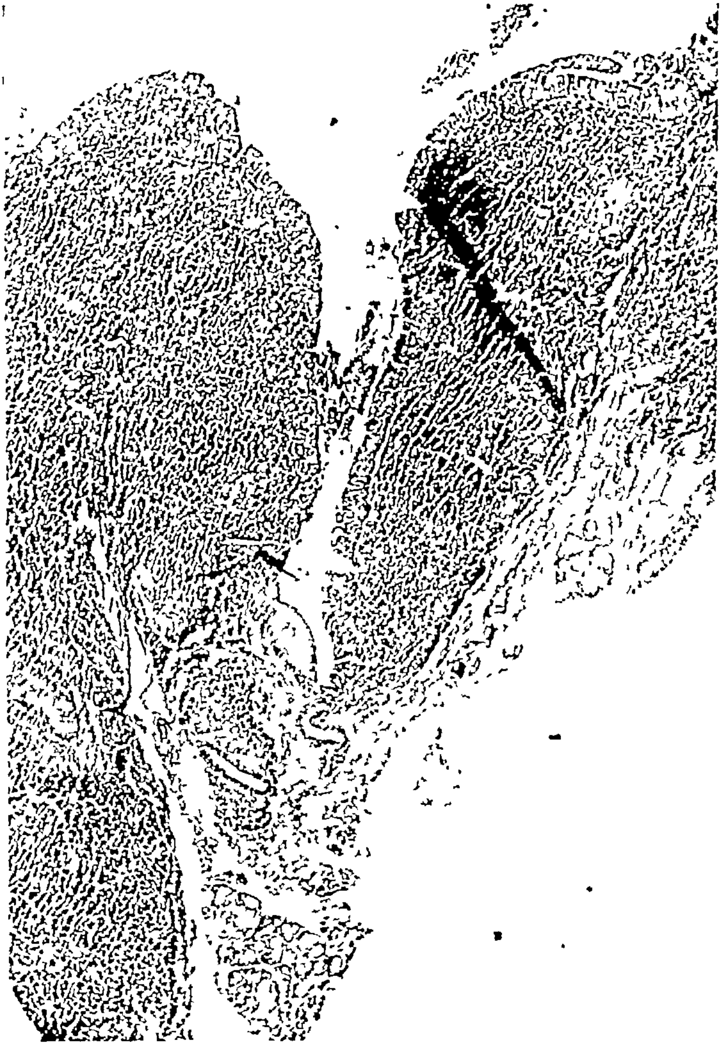


Fig 3 Plica salpingonasalis from radical adenectomy Plica salpingonasalis largely lymphoid tissue, with small portion of glandular elements Squamous metaplasia seen with a duct.

infoldings, which on serial section are seen to be continuous with the gland ducts, give a rugged, slightly nodular appearance to the surface of the folds.

As might be anticipated, none of the specimens presented any acute inflammation. Infrequently, histological evidence of low-grade inflammatory reaction such as focal squamous metaplasia, edema and lymphocytic infiltration of the epithelium, reticulum hyperplasia in the lymphoid aggregates and, rarely, fibrosis were noted. The majority of sections revealed not even this much inflammatory alteration.

In the latter specimens neither voluntary muscle nor cartilage was found, although in the earlier specimens, due to inexperience with the problem, some striated muscle and, once, cartilage, was seen.

CONCLUSIONS.

The literature is replete with articles advising irradiation after adenoidectomy for destruction of the remaining or reformed lymphoid tissue.

In our opinion, many a radium treatment will not be necessary if we remove the hypertrophied folds during adenoidectomy.

It is not at all a rare occurrence that a simple adenoidectomy does not relieve mouth breathing.

We found in our selected series that in 10 cases the enlargement of these folds was so big that the removal was advisable. In two of our cases simple adenoidectomy had been done previously without restoring the nasal breathing. During a subsequent operation no other obstruction was found except for hypertrophied folds originating from the tubal ostium. The removal of these folds restored the free passage of air through the nose.

The elimination of big masses of lymphoid accumulations in a place so openly exposed to bacteria will diminish the number of focal infections.

In this article a radical adenoidectomy is described. This surgical procedure allows the removal of all macroscopically visible lymphoid tissue from the nasopharynx *under visual control.*

SUMMARY.

It is suggested that no adenoidectomy should be considered complete without the examination for removal of diseased or hypertrophied salpingonasal, salpingopalatine and salpingopharyngeal folds.

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LARYNGEAL GRANULOMA FOLLOWING INTRATRACHEAL ANESTHESIA.

A REPORT OF FOUR CASES *

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With the rapid advances being made in surgery, especially relative to the head, neck and thorax, the advantages of endotracheal anesthesia are becoming more and more manifest. Highly skilled surgical and anesthesia teams are of necessity using this form of anesthesia with increasing frequency.

A relatively rare yet significant sequel of prolonged endotracheal anesthesia is nonspecific contact granuloma of the larynx. A review of the available literature reveals that only some 21 cases have been reported since R. J. Clausen's original contribution in 1932[1] Undoubtedly, many more granulomata have been observed at various clinics but have not been published to date.

A review of the literature, including our four cases, can best be presented in chart form.

In all instances reported, the duration of anesthesia varied from 55 minutes to five hours, with the great majority ranging from two to three hours Most of these intubations were performed through a laryngoscope by the anesthetist who noted no existing abnormality. Hoarseness, which is the major complaint referable to granuloma of the vocal cord, appeared as early as 10 days and as late as seven months following operation; however, this symptom became apparent in the majority of cases in one to two months after the intubation Late symptoms may be aphonia and occasionally dyspnea. In 13 instances, the lesion was unilateral and in 12 it

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*From the Otolaryngology Service of Dr Max L. Som.

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was bilateral. On nine occasions the granuloma recurred after laryngoscopic removal.

We believe that the most important factor which may be contributory to the formation of laryngeal granulomata is injury to the mucous membranes. It is only at this anatomical location that these lesions have been described. This trauma to the vocal cords may be of direct or indirect type. The direct type of trauma referred to is that occurring during the actual process of intubation by the anesthetist. This may occur in either the blind or the visual method of insertion of the endotracheal tube. Indirect trauma is that type of injury to the laryngeal mucosa which is caused either by static pressure of the tube *in situ* or by motion of the tube secondary to movement of the larynx and tracheobronchial tree during respiration, coughing, straining, etc. The posterior commissure and vocal processes are the areas where the tube would naturally tend to rest and exert the maximum pressure. The vocal process of the arytenoid is particularly vulnerable to trauma since the cartilage is notably less yielding than muscle and fibrous tissue. Dwyer *et al.*[12] noted that the conventional Magill tube is curved in only one plane. When the tube is inserted into the trachea, the larynx acts as a fulcrum so that the posterior portion of the larynx and the anterior tracheal wall are exposed to constant pressure and trauma. His studies showed more severe congestion of the posterior third of the cords, especially when a large endotracheal tube was used.

It is believed that the initial lesion is a superficial ulceration of the mucous membrane overlying the cartilaginous vocal process. Subsequently, in the attempt at healing, granulation tissue is formed from the submucosa. These granulations must then protrude into the ulcer area which is being encircled by a more slowly growing squamous epithelium. When first seen, such lesions are sessile in character and, due to the differential rate of growth of the tissues, constriction of the base of the granulations occurs, giving rise to a pedunculated mass. In the case reported by Clausen,[1] this pinching off process proceeded to such extent as to allow the granuloma to be coughed up by the patient.

Constitutional factors may possibly make the larynx more vulnerable to trauma.[13] Generalized systemic debility, avitaminosis, anemia, alcoholism and the atrophic mucosa of older individuals may be contributing factors for the lowered resistance to injury by the endotracheal tube.

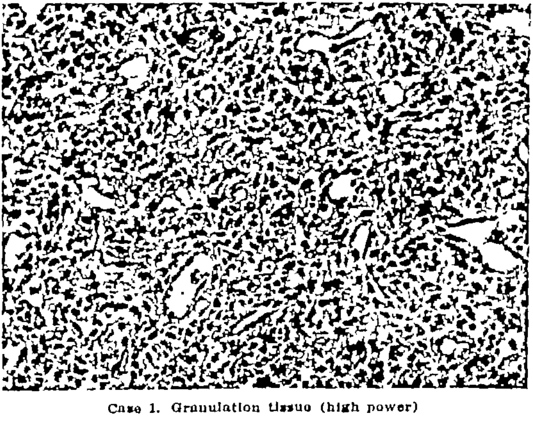
CASE REPORTS.

Case 1: I. C., a 35-year old female, was admitted to the Otolaryngology Service at Beth Israel Hospital on Sept. 20, 1948, with the complaint of hoarseness of three weeks' duration. Mirror examination revealed a rounded pedunculated mass attached to the inferior aspect of the left vocal process. The lesion appeared to be covered by an intact mucous membrane which presented a slightly bluish appearance.

A subtotal thyroidectomy had been performed on this patient about three months previously. Cyclopropane and ether anesthesia was introduced through a nasotracheal Magill tube inserted into the trachea under direct vision. The vocal cords appeared normal at that time. The duration of anesthesia was two hours and 50 minutes.

Postoperatively, hoarseness was noted on the second day and persisted until the sixth day, when it subsided. After a two-month interval, she again developed hoarseness which brought about her admission to the hospital.

Operation: Under direct laryngoscopy with local anesthesia the mass was removed by applying a forceps to its pedicle. The lesion measured about 1 cm. in diameter.



Case 1. Granulation tissue (high power)

Pathology: Granulation tissue with an epithelial surface of stratified squamous epithelium. There were numerous lymphocytes and some polymorphonuclear cells in the submucosal area.

This patient was last seen in January, 1950, about one and one-half years after removal of the granuloma, at which time there was no evidence of recurrence.

Case 2: G. H., a 57-year-old white female, was admitted to the Otolaryngology Service at Beth Israel Hospital on Sept. 19, 1949, with hoarseness of four weeks' duration. Indirect laryngoscopy revealed a pedunculated mass, covered by an intact mucosal surface, arising from the region of the left vocal process.

About seven months prior to this admission, in February, 1949, this patient had undergone a partial colectomy for carcinoma. Endotracheal cyclopropane and ether anesthesia was given throughout a five-hour operation. The vocal cords prior to surgery were normal. There was no postoperative hoarseness.

Operation: Using indirect laryngoscopy with local anesthesia, the mass was removed at its pedicle by a grasping forceps. The tumor measured 8 mm. in diameter and the pathological report was nonspecific granuloma. The patient was again seen on Jan. 9, 1950, at which time she again complained of hoarseness of two weeks' duration. Laryngeal examination revealed a recurrence of the mass over the left vocal process. This tumor was again removed with the indirect laryngeal forceps.

She has been followed in the outpatient department and no evidence of recurrence has been noted to date.

Case 3: M. F., a 70-year-old white male, was admitted to the Otolaryngology Service at the Beth Israel Hospital on Jan. 9, 1950, with the complaints of hoarseness and difficulty in raising sputum, of two weeks' duration. Mirror examination revealed bilateral masses attached to the vocal process of each vocal cord. They were pedunculated and would descend below the cords on inspiration and swing out over the cords on expiration. An intact mucosa covered both tumor masses.

On Nov. 7, 1949, two months previously, a resection of the sigmoid portion of the colon had been performed under endotracheal cyclopropane and ether anesthesia. The intubation was described as being "difficult." No abnormality of the vocal cords was noted preoperatively. Anesthesia lasted two hours and 30 minutes. There was no hoarseness during the postoperative course in the hospital.

Operation: Under indirect laryngoscopy with local anesthesia, both polypoid masses were removed with the laryngeal forceps. Each mass measured about 7 mm. in diameter. The pathological report was nonspecific granuloma.

Postoperatively, this patient has been followed in the outpatient department and showed no evidence of recurrence when last seen on June 14, 1950, five months later.

Case 4: C. C., a 39-year-old white female, was seen in September, 1947, with a history of having had a radical mastectomy performed at another hospital four weeks previously. The duration of the surgery was well over two hours. Anesthesia was endotracheal in type. No complaints were noted in the hospital.

Author	Duration of Anesthesis	Type of Intubation	Onset Symptoms of Granuloma	Unilateral or Bilateral		Recurrence
Clausen	(1) ?	?	8 mos.	X		no
Gould	(2) 5 hrs. 10 min.	blind	2½ mos.	X		yes 2 mos.
Cohen	(3) 55 min.	blind	2½ mos.	X		no
Smiley	(4) 1 hr. 35 min.	direct, larynx normal	10 days	X		yes 2 mos.
Farrior	(5) 3 hrs. 45 min.	direct, larynx normal	14 days	X		no
Kearney	(6) 5 hrs.	direct, larynx normal	1mo.		X	no
Tuft	(7) 5 hrs. 45 min.	blind	1 mo.		X	no
Barton	(8) 5 hrs.	direct, larynx normal	2½ mox.	X		no
Burton	(8) ?	direct, larynx normal	2 mos.	X		no
Finer	(9) 3 hrs. 40 min.	direct	1 mo.	X		no
Hill	(10) 4 hrs.	direct, larynx normal	1 mo.		X	yes,2 mos.
Wattles	(11) 1 hr. 40 min.	Direct, larynx normal	2½ mos.		X	no
New & Devine	(13) not given	not given	not given	2	7	5 patients
		CASES PRESENTED	IN THIS PAPER			
1. C.	2 hrs. 50 min.	direct, larynx normal	2 mos.	X		no
G. H.	5 hrs.	difficult intubation	7 mos.	X		yes, 4 mos.
M. P.	2 hrs. 25 min.	blind difficult	2 mos.		X	no
C. C.	2/ hours	unknown	4 weeks	X		no

Shortly after discharge from the hospital, the patient became hoarse. Mirror examination revealed a mass arising from the right vocal process, covered by an intact mucosa. The tumor almost completely occluded the interarytenoid space.

Under indirect laryngoscopy with local anesthesia, the mass was removed with a grasping forceps. The pathological report was nonspecific granuloma.

The patient was re-examined in January, 1948, and no evidence of recurrence was noted. The patient has had no laryngeal complaints to date (September, 1950).

Case 4. Low power showing granulation tissue and ulcer base.

DISCUSSION.

From a clinical standpoint, the most significant symptom of this condition was hoarseness occurring from one to two months after endotracheal anesthesia. The importance of early diagnosis and prompt removel of the lesion must be stressed. In our review of the literature several cases were noted where treatment was delayed from two to four weeks, and the patients subsequently showed aphonia and marked dyspnea, even to the point of impending asphyxia. These cases were reported by Kearney,[6] Gould[2] and Smiley.[4]

The single curve of the Magill tube, as pointed out by Dwyer *et al.*,[12] may be more of a contributing factor in the etiology of these lesions than is commonly suspected. The modification of the Magill tube to a double curve, simulating the anatomical curvatures as suggested by Dwyer *et al.*, is certainly worthy of consideration as a possible method of preventing these lesions It would seem that the endotracheal tube should be of the smallest size compatible with the lumen of the larynx.

SUMMARY.

1. A review of the literature reveals 21 cases of granuloma of the larynx following endotracheal anesthesia

2. Four additional cases from our clinic are discussed.

3. The etiological factors, pathology, and possible methods of preventing these lesions are also presented.

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77 Park Avenue.

UNIVERSITY OF ILLINOIS INITIATES MODIFIED PROGRAM.

For the period of the national emergency, the University of Illinois College of Medicine will offer a combined three-year residency training program in otolaryngology, which will include the basic course material, in fulfillment of board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved, and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

CORRECTION.

In the October, 1950, issue of THE LARYNGOSCOPE, page 1053, an article entitled, "New Hearing Aid Features World's Tiniest Receiver — Offers Widest Frequency Range, More Overtones," the word advt. (advertisement) was omitted from the end of this article.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will hold its annual meeting at the General Oglethorpe Hotel in Savannah, Ga., on March 2 and 3, 1951.

The lecturers and their subjects are: Dr. A. D. Ruedemann, Detroit, Mich., Cataracts as a Medical Problem and Selection of the Type of Operation in Cataract Surgery; Dr. Peter C. Kronfeld, Chicago, Ill., Diagnosis and Medical Treatment of the Glaucomas and the Surgical Treatment of the Glaucomas; Dr. William C. Owens, Baltimore, Md., Surgical Treatment of Horizontal Muscle Deviations and Surgical Treatment of Vertical Muscle Deviations; Dr. C. Stewart Nash, Rochester, N. Y., Functional Diseases of the Nose and Otolaryngological Mishaps; Dr. Philip Meltzer, Boston, Mass., Simple, Practical Therapeutic Measures in Otologic Practice — Surgical and Non-Surgical, and The Conservation of Hearing in Chronic Suppurative Otitis Media; Dr. Edwin C. Broyles, Baltimore, Md., Tumors of the Larynx — Benign and Malignant — and Summary of Otolaryngological Conditions That Require Expert Attention.

Members and guests are invited to make their reservations directly with the hotel. Registration fee for the lectures is $20.00.

In Memoriam

PERRY GLADSTONE GOLDSMITH, C.B.E., F.R.C.S., M.D., Ch.M.,

1872-1951.

In the passing of Dr. Goldsmith on Jan. 21, 1951, Canada lost perhaps her most distinguished representative of the past half century in the specialty of otolaryngology. As a staunch supporter of the British school, he formed one of the strongest links between this group and that of America, as he always took an active interest in all the senior American societies.

He was born in the small Ontario town of Warkworth, as the son of a medical practitioner and graduated from Trinity College of the University of Toronto. His postgraduate training in otolaryngology was taken in England, where he was house surgeon to such great names as Mr. Dundas Grant and Sir Sinclair Thomson, both of whom he frequently quoted. Before World War I he was attached to the University of Toronto and the Toronto General Hospital, but, as he was actively interested in army affairs, he enlisted and went overseas with the First Canadian Division as a battalion medical officer in 1914. His army career was distinguished throughout and he was the commander of the first Canadian field ambulance sent to France. He had successive posts as commanding officer of various units and was "mentioned in dispatches" in two successive years. In 1918 he was awarded "Commander of the British Empire" (military division), which is a coveted distinction.

Following the war, Dr. Goldsmith resumed his civilian duties and, in 1922, was appointed professor of otolaryngology in the University of Toronto and chief of service of this department in the Toronto General Hospital. He ably filled these appointments until 1939, when he retired to continue private practice until 1950.

Perhap his most useful service to our specialty was his presence as an examiner, for a good many years, on the American Board of Otolaryngology. His wise counsel and his sympathy towards the candidates was an example which was appreciated by all his more junior confrérès. He will be missed from his position as senior counselor on this board.

In addition to being a Fellow of the Royal Society of Medicine of England, he was also a Fellow of all the senior American societies. These included: American Otological Society; American Laryngological Association; American Laryngological, Rhinological and Otological Society, Inc.; American Academy of Ophthalmology and Otolaryngology; American College of Surgeons. He was president of the American Laryngological, Rhinological and Otological Society, Inc., when it met in Toronto, in 1934, and was guest of honor on one occasion of the American Academy of Ophthalmology and Otolaryngology.

It is difficult not to add a personal note to this obituary. Dr. Goldsmith was one of the most ardent readers of the English and American literature of our specialty and also that of general medicine and surgery. He was kindly, helpful and critical of the juniors in his department. An ardent gardener, his rock garden was noted as one of the most outstanding in Canada and he was elected a life member of the Ontario Horticultural Society. His love of the arts was well illustrated by the splendid collection of paintings, china and antique furniture so profusely found in his home. These all showed evidence of a soul much more deeply rooted than that of the average physician or surgeon of our day.

At this point I quote from two letters of appreciation:

"Another tall pine has gone.

"Goldsmith, like Birkett, was a link with old times, and, like him, was a good soldier.

"I used to have good times with him going out to various rock garden specialists. We had a nice trip to Portland gardens when he was last here. He was a real dirt gardener.

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* * * *

The funeral services presented some sadness in that he had been predeceased by his wife and only son so that he left only a sister and a granddaughter behind him. The honorary pallbearers were represented by three emeritus professors from various departments and three active professors. The American Board was represented in this group by Dr. Harris P. Mosher, who had been a personal friend for many years, and by Dr. Percy E. Ireland. It speaks well for the respect in which he was held that such a distinguished group of medical confrérès were present to pay honor at his last rites.

P. E. I.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

Feb. 1, 1951.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac; Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

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P. E. I.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700

Manufacturer: Telex, Inc Minneapolis 1, Minn.

Tonemaster Model Ro[illegible]

Manufacturer: Tonemaster [illegible], 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube [illegible]

Manufacturer: Trimm, I[illegible] Lake St., Libertyville, Ill.

Unex Model "A"; Unex [illegible] Model 95; Unex Midget Model 110.

Manufacturer: Nichols & [illegible], [illegible]thorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co. [illegible] N. Henderson St., Dallas 6, Tex.

Western Electric Model 6[illegible], Western Electric Model 64; Western Electric Models 6[illegible] and 66.

Manufacturer: Western Electric Co. Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp, 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp, Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Palmer House, Chicago, Ill., Oct. 9-12, 1951.

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NASOPHARYNGEAL CANCER.[*][†][‡][§]

DANIEL MILLER, M.D.,
Boston, Mass.

PURPOSE.

From time to time a review of the literature and a re-evaluation of the management of specific disease entities seems necessary. In such a review some phase of the disease such as diagnosis, therapy, racial or geographic distribution is often specifically emphasized. Since one of the distressing features of our subject—nasopharyngeal cancer—is the timelag in arriving at a proper diagnosis (as compared with early diagnosis of cancer in other parts of the body) a desire to reduce this delay has been the stimulating force for our review. It is apparent that early diagnosis in cancer of the nasopharynx will mean more cures in this previously considered hopeless disease.

Although cancer of the nasopharynx is strictly an otolaryngological disease by its primary location, accurate knowledge of its pathogenesis must be considered essential for those in the three major specialties of neurology, ophthalmology and otolaryngology, as well as for the general practitioner. It must

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[†] Pondville Hospital (Massachusetts Department of Public Health). P. O. Walpole, Mass. Reprint number 143.

[‡] Massachusetts General Hospital, Boston, Mass.

[§] Massachusetts Eye and Ear Infirmary, Boston, Mass.

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be emphasized that such knowledge is essential to the general practitioner since the patient for the most part sees his family physician first. Hauser and Brownell[1] stated it well by saying that "nasopharyngeal cancer should be suspected by the general practitioner and diagnosed by the otolaryngologist with ease."

It is alarming to read the literature and discover that so many patients with cancer of the nasopharynx had not had correct diagnoses made until it was too late for cure; and that, in addition, such patients had been subjected to innumerable and unnecessary operations in the belief that their symptoms were due to some other disorder. H. E. Martin and Blady[2] reported that 46 per cent of their patients had received surgical treatment to relieve symptoms before a correct diagnosis was established. Simmons and Ariel[3] reported that 12.7 per cent of their series had been operated upon for symptomatic relief before a correct diagnosis was made. This surgery included tonsillectomies, mastoidectomies, radical sinus operations, dental extractions and many other procedures. In 1931, New[4] stated that 185 operations on 194 patients with malignant lesions of the nasopharynx had been performed for symptomatic relief prior to admission to his clinic; among these were 56 nasal operations, several mastoidectomies, repeated myringotomies, alcohol injection for trifacial neuralgia, and numerous dental extractions. Kleinfeld,[5] in 1936, reported that 15 of his 40 cases had had 20 useless operations, done from one to six months before admission, ranging from craniotomy to radical sinus and mastoid surgery. Needles[6] reported 35 cases treated prior to 1937 with 12 patients having been subjected to unnecessary operations before correct diagnoses were made.

HISTORY.

Although cancer of the nasopharynx is not a very common disease, it is still sufficiently prevalent not to be considered among the diseases rarely encountered. As additional attention is given to this disease more cases will be uncovered. This seems evident in view of the marked increase in the number of cases reported since its first description.

In 1924, Smith and Dawson[7] reported finding erosion of the base of the skull in a Byzantine mummy and they thought it might be due to a nasopharyngeal cancer.

In 1901, Jackson[8] reviewed the European literature, and the earliest case reported was that by Durand-Fardel in 1837. Jackson found 13 cases in all, including one of his own. In 1911, Citelli[9] reported on 10 cases. In the same year, 1911, Trotter[10] described his classical triad for suggesting cancer of the nasopharynx: it consisted of impaired hearing, neuralgia of the second branch of the Vth nerve, and asymmetry of the soft palate. In 1913, Oppikofer[11] reviewed 250 cases with a detailed description of the signs and symptoms of nasopharyngeal cancer. In 1921, Regaud and Reverchon[12] reported on the pathologic features of lymphoepithelioma of the nasopharynx. In the same year, Schminke[13] reported a similar pathologic picture describing this same tumor.

New,[14,15,16,4,17] in 1921, 1922, 1925, 1931 and 1943, has successively reported on the greatest number of cases in the literature to date, and has contributed as much as any individual to the understanding and proper management of the disease.

In 1923, Crowe and Baylor[18] reported 52 cases. A few years later, in 1927, Quick and Cutler[19] first described the transitional cell cancer of the nasopharynx as a histopathologic entity. In 1928, Hansel[20] reported on 12 cases and reviewed the literature as a candidate's thesis for membership in the American Laryngological, Rhinological and Otological Society, Inc., just as the present author is doing 22 years later.

In 1929, Ewing[21] made the major contribution of a report on 110 cases with excellent histopathological differentiation.

In the 1930's, Dunlap,[22] C. L. Martin,[23] Furstenberg,[24] Kleinfeld,[5] Kasabach,[25] Hauser and Brownell,[1] Needles,[6] Digby,[26,27] and numerous others[28-36] began to fill the literature with various reports of cases.

In the 1940's, the outstanding papers among many .were those of C. L. Martin,[37] H. E. Martin and Blady,[2] New and

Stevenson,[17] and Simmons and Ariel,[3] the latter being the most recent comprehensive report of 150 cases.

The author's present series includes patients treated at the Massachusetts General Hospital, Massachusetts Eye and Ear Infirmary, and Pondville State Hospital from 1930 through 1947; also patients treated at the Collis P. Huntington Memorial Hospital from 1930 until the hospital closed on Jan. 1, 1942. This series includes 102 cases.

ANATOMY INCLUDING LYMPHATIC DRAINAGE.

In order to appreciate fully the significance of numerous signs and symptoms that may suggest nasopharyngeal cancer, an anatomical perspective must be achieved.

Briefly, the nasopharynx may be described as a six-walled compartment. Its "anterior wall" is composed of the posterior portion of the nose, including the septum and the choanae through which the posterior tips of the middle and inferior turbinates project. One might also consider that it includes a portion of the soft palate along its inferior margin. The "floor" is the opening into the oropharynx, but on swallowing, the soft palate sweeps back to the posterior pharyngeal wall to form such a true floor. On the "posterior wall' the mucosa covers the fascial attachment of the superior constrictor muscle as it lies over the anterior portion of the basilar process of the occipital bone where it joins the sphenoid bone. On the "roof" of the nasopharyngeal cavity, the mucosal wall covers the main body of the sphenoid bone. The "lateral walls" are penetrated by the Eustachian tubes; surrounding the tubal orifice in a posterosuperior plane is the rounded eminence called the torus tubarius. The fossa formed by the torus and the actual lateral wall is the well known fossa of Rosenmüller, a very common site for the origin of nasopharyngeal cancer. The adenoid usually begins centrally on the posterior wall and extends up to and includes a portion of the superior wall, or the roof. An important adjacent structure is the foramen lacerum through which the internal carotid artery passes into the skull. The foramen lacerum is located about 1 cm. anterior to the fossa of Rosenmüller and has been repeatedly shown to be a direct pathway for the local extension of tumor into the intracranial cavity; also, *about 1 cm. lateral to the foramen lacerum are the foramen* ovale and the foramen spinosum where still further direct intracranial extension can occur.

The mucosa covering the nasopharynx is composed of pseudostratified ciliated columnar epithelium which changes to stratified squamous epithelium as it approaches the oropharynx. There are occasional collections of mixed serous and mucoid secreting gland cells from which the occasional adenocarcinoma or even mixed tumor may arise. Beneath the *epithelial layer in the corium are numerous lymphoid follicles* from which small efferent ducts evolve.

Since in so great a number of cases cervical metastasis is the presenting symptom, the knowledge of lymphatic drainage of the nasopharynx is of importance. The lymphatic ducts begin in the adenoid and lymphoid tissue of the nasopharynx. Some of these join and descend inferiorly to the lateral and medial retropharyngeal nodes. Others pass laterally directly into the neck and to the upper deep cervical glands which are located just inferior to the superior attachment of the sternomastoid muscle. Some of the efferents from the retropharyngeal nodes also often extend to the deep cervicals. Various combinations may, of course, occur. Any of these efferents may then descend along the internal jugular vein where they form the jugular chain of nodes, and these in turn may connect with the supraclavicular region where is found the supraclavicular *group of nodes. Efferents from the nodes of the* upper deep cervical glands run along the anterior border of the trapezius muscle, following the course of the spinal accessory nerve, enter and then leave this so-called spinal accessory group of nodes to join with the supraclavicular nodes.

Thus the important lymph node groups are: *1.* the retropharyngeal nodes, medial and lateral; *2.* the upper deep cervicals; *3.* the internal jugular chain; *4.* the spinal accessory group; and *5.* the supraclavicular group.

HISTOPATHOLOGY.

Nasopharyngeal tumors as a group are just as varied as tumors in other parts of the body, but specific types of neoplasm are more prone to develop in the nasopharynx. As a general rule it might be said that most tumors of the nasopharynx are malignant. Stout[38] found 34 different tumor types in a review of the literature, including those in his own series. He reported from the records of the Presbyterian Hospital, of New York City, on 82 cases of nasopharyngeal tumors, of which 23 were benign and 59, or 72 per cent, malignant.

Among the primary malignant tumors of the nasopharynx in order of frequency Stout[38] found various grades of differentiation of epithelioma the most common, then lymphoepithelioma, lymphosarcoma, occasional plasmocytoma, fibrosarcoma and malignant teratoma. Among the secondary malignant tumors he mentions chordoma, craniopharyngeal duct tumors, leucemia, Hodgkin's disease and Boeck's sarcoid.

There has been considerable confusion throughout the literature regarding the nomenclature of the nasopharyngeal neoplasms which persists today in the various medical centers. As tumors of varying terminology with similar descriptions are placed before the otolaryngologist he must accept them as such for the present. Because of this problem, Salinger and Pearlman,[39] in 1939, in a report of 24 cases stressed the variation in the histopathology. They had three pathologists review all of their cases. They found the so-called transitional cell carcinoma as described by Quick and Cutler[10] as predominant in 75 per cent of their series; lymphoepithelioma was diagnosed in only six of their cases; however, pathologists still change the terminology and 15 years after their report confusion still abounds.

F. W. Stewart has been quoted by H. E. Martin and Blady[2] as employing the following basic differentiation of the epithelial tumors of the nasopharynx: the term squamous carcinoma includes those tumors with definite keratinizing tendencies; the transitional cell carcinoma included the type in vhich the picture suggests duplication of the intermediate ayer of the stratified epithelium. The term lymphoepitheli-ma is more difficult to differentiate because many of these umors are really lymphosarcomas and are usually those in vhich the epithelial elements and the lymphoid elements have oth undergone anaplastic change.

In our series the following histopathologic diagnoses were eported *by the pathologists in the institutions from which* ur cases are recorded:

DIAGNOSIS BREAKDOWN.

	Total	Five-Year Survivals
Epidermoid carcinoma	49	1
Lymphoepithelioma	8	1
Undifferentiated carcinoma	4	1
Lymphoma	5	1
Plasmocytoma	4	—
Reticulum cell sarcoma	2	1
Adenocarcinoma	1	—
Lymphosarcoma	1	1
Transitional cell carcinoma	17	3
Embryonal carcinoma	1	—
Carcinoma (unclassified)	2	—
Carcinoma (clinical diagnosis)	8	2
	102	11

The above chart demonstrates the great variety of histopathologic reports and why C. L. Martin,[33] in 1939, stated that "fortunately all the malignant neoplasms in the pharynx are very radiosensitive and their histopathology is not of any great clinical importance." *In 1943, he qualified this*[34] *and* stated that "85 per cent of the nasopharyngeal malignancies fall into the radiosensitive group including undifferentiated epithelioma, lymphoepithelioma, lymphosarcoma and plasmocytoma. Adenocarcinoma and well-differentiated epithelioma, spindle cell cancer and the more differentiated chordoma, respond poorly to the doses of radiation usually effective in the former group."

Two tumor types found only occasionally in the nasopharynx deserve a more detailed description of their histology and life history. One, the chordoma, has already been well described by Furstenberg.[21] This is a tumor which has its origin in the remaining notochordal cell. It is most common at either extremity of the spinal column and arises in the vertebrae. Furstenberg reviewed the literature and found 52 cases reported, of which only 20 were located in the region of the nasopharynx. These tumors are of low malignancy but because of their location can rarely be removed surgically due to obvious technical difficulty.

The other tumor, the so-called nasopharyngeal fibroma, has also been well described in the past. It is an "adolescent" tumor occurring only between the ages of five and 25, after which it tends to disappear spontaneously; however, by its location it can act as a malignant lesion because of the pressure caused by its expansion. It is a smooth, solid tumor which is composed of fibroblasts, reticulum fibres which give it a firmer consistency than the true choanal polyp, and it contains a great many capillary blood vessels. It is highly vascular and even a biopsy may result in serious hemorrhage. The present treatment consisting of electrosurgical removal followed by radon seed implantation has been found extremely effective. The author has noted 11 cases in a 17-year review; all of these patients are alive and well and free of the tumor. These cases ranged from 11 to 21 years of age, there being 10 males and one female.

Plasmocytoma is considered of doubtful malignancy by Stout.[38] It tends to recur locally and often cannot be distinguished from the multiple myeloma of the plasma cell type. Malignant teratoma and dermoid cysts appear at birth or shortly thereafter — a good diagnostic point — and are very rarely found in the nasopharynx.

INCIDENCE: AGE, SEX AND RACE.

Nasopharyngeal cancer is not very common, but it occurs often enough, and its early diagnosis is made infrequently enough to warrant its emphasis. Nasopharyngeal cancer has been reported by Hertzfeld, according to Lack,[40] in 1906, as one case in 28,000 cases of cancer. He also stated that Finder calculated five cases in 40,000 and Gurlt, 18 cases in 10,000.

H. E. Martin and Blady[2] reported 2 per cent of all the malignant cases in the head and neck clinic at the Memorial Hospital in New York City to be nasopharyngeal cancers. At Pondville State Hospital, from 1930 to 1947, among a total of 13,578 malignancies, 27 were nasopharyngeal cancers, or an incidence of 0.002 per cent.

In the author's series the average as well as the median age is 49. The youngest case was age five, the eldest, 86. There are 20 patients under 40 years of age, 53 patients between 40 and 59 years, and 27 patients 60 years or over. In two cases the age is unknown or unstated.

The majority of nasopharyngeal tumors in our series as well as in others was found in males, the ratio being 76 males to 26 females.

In the past two decades mention has been made of the frequency of nasopharyngeal cancer in the Chinese. Digby, Thomas and Tse,[24] in 1930, in an outstanding article with detailed anatomic and histopathologic sketches, reported on 103 cases. They stated that in their 42-bed service in a Hong-Kong hospital it was not unusual to have four or five cases of cancer of the nasopharynx at one time. Dunlap,[25] in 1938, reported having treated 16 patients in six years of private practice, all of them either Chinese or other Orientals. He made mention of the fact that 40 per cent of his private patients were Chinese. He reported that Dr. Lois Green "writes from Changsha that one of her students found that the incidence of nasopharyngeal tumors there is 16 out of 336 cases." In our series it is of interest to note that there are four Chinese patients, or 3.9 per cent of those reported.

PATHOGENESIS AND SYMPTOMATOLOGY.

In view of the varied clinical findings and actual structural involvement, the resulting symptoms of the disease must be considered in some detail. It is in this particular area of the problem of cancer of the nasopharynx that the clinician must be well grounded, whether he be the internist, general practitioner, ophthalmologist, neurologist or otolaryngologist.

The diagnosis may be difficult to establish on first examination for many reasons. It may be nonapparent grossly on close, careful scrutiny; it may be represented as a slight fissure in the fossa of Rosenmüller; the first awareness of its presence may be the clinical finding of upper deep cervical lymphadenopathy. The latter is usually investigated by biopsy excision and may be found to contain transitional cell cancer or lymphoepithelioma, typical of a primary lesion in the nasopharynx; even then, repeated study of the nasopharyngeal vault might show no gross pathology. Often, following the initiation of radiation therapy to the glands, including the nasopharynx in the field, there will occur a breakdown in the superficial mucosa of the nasopharynx and the submucosal tumor then may be seen for the first time.

As the primary lesion enlarges in the nasopharynx it may encroach upon the Eustachian tubal orifice and produce unilateral blocked feeling in the ear, tinnitus, sensation of fluid in the ear, decreased hearing, and occasionally slight dizziness. At this time the drum is usually retracted. Purple to clear yellow serum may be noted through the drum in the middle ear, and the hearing loss is of a conduction type. Persistent repeated secretory otitis media may suggest the presence of a nasopharyngeal cancer involving the Eustachian tube.

The tumor as it enlarges may encroach upon the soft palate and produce a "nasal" voice. Increased postnasal discharge, occasionally bloody, soon ensues. After the tumor extends to the choanae, unilateral nasal obstruction and increased nasal discharge, occasionally bloody, takes place. Repeated epistaxis may occur as a single symptom. If secondary infection occurs at this time a unilateral pansinusitis with its associated symptoms may develop.

The tumor may extend inferiorly and involve the lateral and medial retropharyngeal nodes, producing a lumpy feeling in the upper portion of the throat, associated with some soreness and even pain behind the palate, with referred pain to the ear on that side.

The tumor may then extend anterolaterally and involve the foramen lacerum and then continue on intracranially, pushing the dura ahead of it, actually (though rarely) penetrating the dura only when infection or trauma are superimposed. Marked headache may now become a symptom. The VIth nerve passes directly over the foramen lacerum and is frequently the first cranial motor nerve involved. The IIIrd, IVth and Vth cranial nerves lie just above the VIth nerve and as a result are the next to be involved. This situation would produce diplopia, due to internal strabismus, and parasthesias and pain along the branches of the Vth nerve, such as in the teeth, cheek and nose. As the sympathetic nerve chain about the internal carotid artery in the foramen lacerum is involved, Horner's syndrome with its miosis and enophthalmos occurs. Involvement of the superior cervical ganglion in the neck by direct extension from the internal jugular chain of involved cervical nodes can produce this same syndrome. The tumor may then extend from the foramen lacerum to the carotid groove and then to the supraorbital fissure causing exophthalmos, more headache, possible vomiting, choked discs, possible optic nerve atrophy, and finally complete ophthalmoplegia may occur.

Schlivek,[11] in 1937, stressed the ophthalmic symptoms he noted in a series of 38 cases. He emphasized that "one can evolve an ocular syndrome of nasopharyngeal tumor: involvement of the Vth, sympathetic, and VIth nerves, either individually or together, with symptoms referable to the ear, are strongly suggestive of a nasopharyngeal tumor." Of his 38 cases, 16 had symptoms referable to the eyes. It can be said that once ocular signs occur, the tumor has already extended intracranially except in the case of Horner's syndrome, which is produced by involvement in the neck.

The tumor may extend laterally and anteriorly from the nasopharynx, entering the Eustachian tube to extend to the middle ear. Mastoiditis has occurred from superimposed infection and operations have been done on the mastoid on numerous occasions without the operator even being aware of the presence of a primary tumor in the nasopharynx. Deafness, middle ear discharge, often bloody, otalgia, and a pulsating tinnitus may develop from this extension of the tumor.

If the tumor should infiltrate along the pterygoid process and involve the internal pterygoid muscle, severe trismus occurs. In such a case described by Moyle[42] this was the presenting complaint of a 21-year-old man.

When the tumor extends anterolaterally and directly involves the levator palatini muscle an asymmetry of the soft palate is produced. Difficulty in swallowing and nasal voice may develop. A bulging of the palate should certainly strongly suggest this lesion.

Occasional extension through the bone of the base of the skull does develop but is less usual than extension via the natural foramina. Bach *et al*[43] reported a case with true extension through the sphenoid bone that continued superiorly and posteriorly to the petrous tip and eventually laterally to involve the internal carotid artery with perforation and fatal nasopharyngeal hemorrhage. True extension can result from stripping of the nutrient periosteum by the tumor, followed by bone infiltration *per se* by the tumor. Simmons and Ariel[3] observed this in six postmortem cases.

In 1942, Lenz[44] reported on a series of bone involvements demonstrated by X-ray. He found the tumor had involved the body of the sphenoid in 10 instances, the greater wing of the sphenoid in 12, the petrous tip in 10, the basiocciput in eight, the foramen ovale and spinosum in five, the posterior margin of the vomer in three, and the sella turcica in three.

At one time or another all of the cranial nerves have been reported to be involved by this disease, but the VIIIth nerve and 1st nerve are by far the rarest. Martin and Blady[3] report the most common order of nerve involvement as the VIth, IIIrd, IVth, Vth, VIIth and IInd. The IXth, Xth, XIth and XIIth cranial nerves may become involved, mainly by extension from the metastatic glands in the neck. If the upper deep cervical nodes enlarge and become confluent with the upper group of the internal jugular chain, extension may take place up to the jugular bulb and then involve the cranial nerves that come through the jugular and hypoglossal foramina, namely, the IXth, Xth, XIth and XIIth. The jugular bulb syndrome of Jackson develops, producing hoarseness due to vocal cord paralysis, palatal paralysis and nasal regurgitation of food, a unilateral paresis and deviation of the tongue, and even some shoulder drop.

Ballenger[48] reported a case which produced VIIIth nerve paralysis by true extension to the petrous with encroachment by the tumor on the internal auditory meatus and involvement and destruction of the VIIth and VIIIth cranial nerves.

The VIIth nerve is more often involved peripherally by the same process that extends from the metastatic glands to the jugular bulb, destroying the nerve near its exit from the stylomastoid foramen.

One can readily see the importance to the neurologist of the nasopharyngeal cancer syndrome in relation to cranial nerve involvement. In 1941, Brock[49] stated that "from the neurological viewpoint a clinical picture referable to unilateral implication of the cranial nerves issuing from the middle or posterior fossa, should make one search carefully for enlarged retropharyngeal or cervical glands, followed by repeat exami-

The chief or initial complaints of the patients in our series in the order of significance to the patient may be listed as follows:

CHIEF COMPLAINT
(Signs or symptoms which brought patient to hospital)

Symptoms	No. of patients
Nasal obstruction	28
Cervical adenopathy	23
Deafness	22
Headache	17
Nasal bleeding	14
Postnasal drip	12
Sore throat	12
Facial pain	8
Dyspnea	6
Neurological symptoms	6
Ear pain	4
Tinnitus	4
Oral bleeding	3
Diplopia	2
Weight loss	2
Local numbness	1
Esotropia	
Ocular protrusion	
Hoarseness	
Cough	

OTHER SYMPTOMS
(Accompanying symptoms of less concern to patient)

Symptoms	No. of patients
Nasal obstruction	15
Weight loss	14
Nasal bleeding	12
Deafness	11
Postnasal drip	11
Headache	10
Tinnitus	7
Dyspnea	7
Sore throat	6
Cervical adenopathy	6
Ear pain	5
Malaise	3
Neurological symptoms	3
Hemoptysis	3
Facial pain	3
Diplopia	2
Esotropia	1
Ocular protrusion	
Blindness	

In this specific disease, unfortunately, the sign or symptom first noted by patient or physician may not appear until the disease has reached a practically incurable stage, with present methods of therapy.

The delay in diagnosis must be overcome. This must be stressed again and again or the five-year cure rate will never be increased beyond its present low level.

The literature is filled with discouraging reports of delayed diagnoses. In 1922, Woltman[48] observed that only one out of 25 cases had been correctly diagnosed before admission to the Mayo Clinic. New,[15] in 1922, reported 21 out of 79 with symptoms referable to the eye, 29 with symptoms referable to the ear, 38 with symptoms referable to the nose or nasopharynx, and 51 with cervical adenopathy. In reporting his

194 cases in 1931, New[4] reported that 42 per cent of these patients had cervical lymphadenopathy on admission.

In 1936, Salinger and Pearlman[13] reported that about 80 per cent of their patients showed metastases on admission. In 1938, Furstenberg[14] reported that 60 per cent of his series of 40 cases had cervical metastases on admission. This contributed a great deal no doubt to his pessimistic attitude regarding the disease which led to his much quoted statement at that time, "My meager experience with 40 cases leads me to conclude that treatment is exceedingly futile and the prognosis hopelessly discouraging." Only one of his 40 patients was alive after two years. Most of his patients came to him 15 months after the initial sign or symptom was observed.

In 1941, Kasabach[15] reported on 57 patients from the Presbyterian Hospital, of New York City, and stated that 40 cases, or 77 per cent, showed cervical adenopathy on admission, and 30 per cent revealed X-ray evidence of invasion of the basilar bones and adjacent bony structures.

As recently as 1949, Simmons and Ariel[3] demonstrated statistically the worst aspect of present day delay and inaccuracy in the diagnosis of cancer of the nasopharynx in patients arriving at their hospital. In their 150 cases, 44 patients, or 29.3 per cent, had involvement of one or more cranial nerves on admission; 130 cases, or 86.6 per cent, had cervical metastases and of these 67, or 44.7 per cent, had bilateral cervical metastases. Of the 150 cases the correct diagnosis was not suspected by the referring physician in 92 cases, or 61.3 per cent. What is most revealing in their stimulating report is that the interval between the onset of symptoms and seeking of medical advice by the patient averaged 3.2 months, whereas there was an average lapse of 6.9 months between the first visit to the physician and the time the correct diagnosis was established. The element of time lag was present to a lesser degree in our reported series of 102 cases.

In further evaluating our series the following accepted stage classification was used:

TOTAL NUMBER OF PATIENTS, 102.

		No. of patients
Stage 1A	(Primary case, without metastases when first seen here)	40
Stage 1B	(Primary case, with metastases when first seen here)	44
Stage 2A	(Secondary case, without metastases when first seen here)	1
Stage 2B	(Secondary case, with metastases when first seen here)	17

Of this group of cases, 61 out of 102, or 59.8 per cent, had metastases when they reported for treatment.

The average duration of symptoms before the first examination was as follows:

AVERAGE DURATION OF SYMPTOMS BEFORE FIRST EXAMINATION.

STAGE 1A (2 unknown)	STAGE 1B (5 unknown)
5½ months	8 months

ALL PATIENTS IN ALL GROUPS

Average duration	9 months (8 unknown)
Shortest duration	1 month
Longest duration	4 years, 5 months

The *delay in treatment* was considered the lapse of time between the date of the first examination and the date on which an accurate diagnosis was made and the treatment inaugurated. A lapse of two months or less was not considered a delay.

In 14 of the 102 patients, treatment for cancer was not begun within two months of the first visit because accurate diagnoses had not yet been made. The average "delay" of these 14 patients was five and one-half months; the longest "delay" was one year and two months; the shortest "delay" was two months and one week.

Early diagnosis is of major importance in preventing further delay in treatment.

TREATMENT.

The treatment of neoplasms of the nasopharynx seems to be less controversial than that in many other forms of cancer.

Since most of the malignancies of the nasopharynx are radiosensitive, radiation has proved to be the treatment of choice.

The results of such treatment are directly related to the time-lag or "delay" in arriving at a correct diagnosis. Since the inception of its use in nasopharyngeal cancer the results of radiation therapy have varied. It is not the purpose of this reviewer to delve into a field that belongs to the much more capable and experienced radiation therapists; however, some discussion is indicated in order to interpret and evaluate the progress made in the treatment of the disease in the past two decades.

Originally attempts were made to eradicate the disease by electrosurgery. For example, in 1930, Digby, Thomas and Tse,[14] reporting on their therapy in the surgical unit of the Hong Kong Hospital, stated that all cases perished despite local "diathermosurgery." No deep X-ray was available to them at the time of that report. In a later article the same year, Digby[27] states "that after ligature of the left common and external carotid arteries, and excision of the left superior maxilla, the growth in the nasopharynx was destroyed by diathermy; and one week later 2,706 mg. hours of radium were applied. Two weeks later 3,102 mg. hours of radium were again administered." All radium was given in 6 mg. hour tubes. He foresaw some hope for the future with the use of radium. In his prophecy he was correct, for not long after occasional reports of five-year cures by men in various parts of the world began to trickle into the literature. As the radiation technique, mainly by external maneuvers, improved, a greater, but not remarkable, number of cures were reported.

The author's series of 102 patients were treated as a general rule in the following manner: For the most part the lesions were treated through lateral pharyngeal portals, cheek portals and occasional intraoral portals. M. Schulz[48] recommends a tumor dose calculated to a minimum of 5,000 r. as sufficient for the usually radiosensitive nasopharyngeal cancers. The patients in this series included many treated in the early 1930's, at which time as little as 3,000 r. tumor dose was the prevalent treatment. The more recently treated patients received a primary course of at least 5,000 r. calculated tumor dose.

In 1938, Blady[50] described a special instrument for irradiation of neoplasms of the nasopharynx with radium directly applied to the tumor, which has been used in several medical centers since then, but not in our particular series. He has been of the opinion, as have several other radiation therapists, that "by a combination of external and intracavitary irradiation, which is less than the maximum doses of either, the desired degree of radiation effect may be produced in the nasopharynx."

Cutler and Buschke,[51] in 1939, advised doses of 7,000 to 8,000 r. (measured on the skin) for carcinoma or lymphoepithelioma of the nasopharynx, while recommending a total dose of about 6,000 r. for lymphosarcoma of the nasopharynx.

As has been stated before, details relating to portals, such as the intraoral approach, or lateral, anterior or posterior portals, as indicated, should be left in the hands of the radiologist who should work in conjunction with the otolaryngologist in order that the maximum effective tumor dose be directed to that area which contains the tumor itself.

Protection of the eyes during this therapy is an important factor and is provided for by the radiation therapist. Of great importance in our clinics has been the increasing attention directed to the teeth, whether they be in excellent or poor condition. It is our policy at this time to recommend the removal of all teeth in the proposed field of radiation regardless of their condition or the age of the patient, at least one week prior to the inauguration of radiation. The incidence of radiation osseonecrosis has been greatly reduced in our experience because of this precaution. This is especially valuable in the cases in which the intraoral portal is to be utilized.

Up to the 1920's practically all patients with nasopharyngeal cancer died. Coutard[52] did a considerable amount of pioneer work in improving the techniques of radiation therapy. *His* results were compiled by *DelRegato* from L'Institute du Radium (1919-1931). Nine cases of carcinoma of the nasopharynx were treated and one case was well, after five years. In 1936, Kleinfeld[5] reported on 40 cases. All of these had died but two, who were but one year past treatment. The longest any of his cases lived was three years.

In 1936, *Duhig*,[31] of *Australia*, reported on the treatment of a 13-year-old girl with cervical metastases. He had achieved only palliative help with radium but was encouraged by its use.

In 1938, Furstenberg[24] reported that only one of his 42 cases was alive after two years. He was certainly discouraged at that time.

The same year, 1938, Hauser and Bramwell[1] reported only one out of their 50 cases was alive five years after treatment.

In 1940, H. E. Martin and Blady[2] showed a great change in the end-results. They reported the tremendous number of 20 five-year cures, or 25 per cent, in a series of 80 patients. The following year, in 1941, Figi[33] reported a 10-year cure in a child with highly malignant cancer of the nasopharynx with bilateral cervical metastases.

That same year, 1941, Kasabach[25] reported 14 cases (33 per cent) of 42 cases living three or more years, but only seven cases (16.6 per cent) of five-year cures.

In 1942, Lenz[44] reported a five-year cure in 27.6 per cent of a series of 44 cases. This report included all cases regardless of the stage of the disease.

New and Stevenson,[17] in 1943, reported on their extensive series of 271 cases, but these included cases treated when radiation therapy for these tumors was given in smaller doses than recommended today; in fact, all these cases were treated prior to 1937. They reported that out of 234 cases with positive biopsies, only 13 per cent were living after five years.

In 1949, Simmons and Ariel[3] reported the death of 71 out of 78 determinate patients, a net five-year survival rate of 9.8 per cent.

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Up to the 1920's practically all patients with nasopharyngeal cancer died. Coutard[52] did a considerable amount of pioneer work in improving the techniques of radiation ther-

It is of striking importance prognostically from the results of our series, that if a patient with a nasopharyngeal cancer in Stage 1A, who has been "adequately" treated, should reach the three-year limit without recurrence, his chances for survival would then be excellent. The 10 three-year cures were also the 10 five-year cures. It is interesting to go back to a statement by Munro Black, quoted by Capps,[54] in 1939. Black,[55] in a Hunterian Lecture, stated that "if a good local result is obtained and the patient passes the three-year limit without distant metastasis, the outlook would appear good. If treated, or only treated late in the disease, most seem to run a fatal course in under two years." The author's statistical survey appears to bear this out.

LOCAL RECURRENCE AND METASTASES.

Cases which recur after primary treatment must still be managed by the physician. The question of more palliative radiation or surgical intervention must be considered from time to time. Neurosurgical procedures for relief of pain have been effective in many cases.

In this series the following time intervals are of interest in relation to recurrence in the various stages:

LOCAL RECURRENCE (Average time lapse after initial treatment)

Stage 1A	1 year, 5 months
Stage 1B	8 months
Stage 2A and 2B	1 year, 6 months

LOCAL EXTENSION (Average time lapse after onset of symptoms)

Stage 1A	6¼ months
Stage 1B	1¼ months

REGIONAL GLANDS (Average time lapse after onset of symptoms)

Stage 1A	*2 years, 1 month*
Stage 1B	3 years, 6 months

Distant metastases seem more common in nasopharyngeal cancer than in other respiratory tract cancer.

DISTANT METASTASES (Average time lapse after onset of symptoms)
(Lungs, axilla, liver, spleen, cecum and bones)

Stage 1A	2 years, 3 months
Stage 1B	10 months
Stage 2A and 2B	2 years, 7 months

C. L. Martin[23] states that "generalized metastases appear in the chest, bones, liver, kidneys, spleen, and retroperitoneal nodes as in any type of disseminated malignant disease." H. E. Martin and Blady[2] reported dissemination of tumor below the clavicle in 34 per cent of their cases. In their series, in those cases with uncontrollable disease the average length of life was about 14 months.

In the 37 autopsies obtained from 128 deaths reported by Simmons and Ariel,[3] 20 patients, or 54 per cent, had generalized metastases.

The nasopharynx itself is rarely the site of distant metastasis. This occurred in a patient treated at the Massachusetts Eye and Ear Infirmary in whom metastasis was found in the nasopharynx from a primary neoplasm of the prostate.

SUMMARY.

A report of 102 cases of nasopharyngeal cancer has been given. These patients were seen and treated at the Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital, Pondville State Hospital, and the C. P. Huntington Memorial Hospital before it was closed.

Of the 102 cases, there were 35 "potential survivors" — Stage 1A (primary case without metastasis when first seen) and 10 of these cases survived three years; and the same 10, or 28.6 per cent, survived five years without recurrence; therefore, all cases in this series that survived three years also survived five years.

In Stage 1B (primary case with metastasis when first seen here) there was but one five-year cure, or 3.1 per cent, of 32 cases.

All stages (1A, 1B, 2A, 2B) included 83 "potential survivors." (Nineteen of the total have been treated in the past three years and, therefore, cannot be included in the five-year "potential survivor" group.) Of these, only 11, or 13.3 per cent, of the entire series are five-year survivors.

The average duration of symptoms before the first examination in Stage 1A was five and one-half months, and in Stage 1B, eight months.

The five most common chief complaints of patients on admission were nasal obstruction, cervical adenopathy, deafness, headache and nasal bleeding.

Fourteen of these 102 patients were not treated for this disease within two months of the first visit because accurate diagnoses had not been made. (The lapse of time between the date when the patient was first examined and the date on which accurate diagnosis was made and treatment, therefore, inaugurated was considered as the "delay.") A lapse of two months was not considered "delay." The average "delay" in these 14 patients was five and one-half months.

CONCLUSIONS.

The results obtained in this series compare favorably with those of other reported series to date.

The problem of early diagnosis can be solved partially by more careful attention to the early signs and symptoms produced by the disease. The time-lag between onset of symptoms and accurate diagnosis must be shortened in order to give the patient a better chance for survival with the present radiation therapeutic techniques.

New's[14] statement of 1922 bears requoting 37 years later. "Malignant tumor of the nasopharynx is much more common than believed. The syndrome which it presents is not generally known. This accounts for the fact that many patients are treated medically and surgically without discovery of the tumor. There is a striking lack of nasal or nasopharyngeal symptoms in many of the cases."

The progress made during the last decade has proven that there is hope for combating this serious disease. Earlier diagnoses mean more survivors of nasopharyngeal cancer.

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THE PERMANENTLY PATENT FENESTRA NOV-OVALIS.*

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Osteogenetic closure of the newly created fenestra following fenestration of the vestibular labyrinth for clinical otosclerosis has been the greatest deterrent to the permanent maintenance of the early hearing improvement obtained following this surgery. This has been recognized by otologists ever since this surgery has come into existence. This fact, though recognizable, was not surprising, since it was natural to expect nature to carry out its usual function of repair within the bony labyrinthine capsule just as it was expected to repair all the other surgically injured soft tissues.

We could hardly have expected nature to repair all injured tissue without repairing the newly created fenestra as well, since the identically existing constitutional biologic factors which govern repair following surgical injury in one type of tissue govern all types of injured tissue.

In recognition of this natural law of overall repair, in order to assure the permanent patency of the newly created fenestra, we were for the first time confronted with the problem of experimentally seeking means and ways of preventing nature from performing its naturally inherent duty of repair in the bony fenestra region without interfering with its mission of repairing the rest of the injured tissues.

With this mission in mind I decided first to observe and study how nature repairs the newly created fenestra, how it

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closes it by osteogenesis and where the osteogenesis starts and ends in a postfenestrated ear. I hoped thus, as a result of the knowledge gained from such a study, perhaps to be able to find ways and means of interfering with nature's mission of repair and preventing osteogenesis either from starting or at least from completing its mission if its start could not be prevented. In all surgery nature's law of repair is the greatest asset to the surgeon, but in fenestration surgery it is both the combined friend and enemy of the otologic surgeon. Here the surgeon expects the unnatural, he wants repair everywhere except within a bony area 2 mm. wide and 6 mm. long which is the fenestra gap created in the bony vestibular labyrinth. Isn't this asking too much of nature?

What have I learned from the above mentioned study and how was this knowledge applied in an attempt to try and serve our purpose of keeping osteogenesis from taking place? I have revised 517 osteogenetically closed postoperative labyrinthine fenestras and studied the observations made in these fenestras. I learned therefrom:

1. That bone-dust when left in the region of the fenestra stimulates and hastens its closure by osteogenesis. I, therefore, advocate that all bone-dust be meticulously removed.[1]

2. That the most meticulous removal of bone-dust does not prevent ultimate bony closure of the fenestra. Fenestras created without the burr and, therefore, without formation of bone-dust, closed as a result of osteogenesis as well.

3. That osteogenesis does not as a rule take place underneath the tympanomeatal flap on the outer surface of the bony labyrinthine capsule in the region surrounding the newly created labyrinthine fenestra.

4. That osteogenesis takes place underneath the tympanomeatal flap as a rule only intramarginally within the fenestra gap in the fenestra rim.

5. That osteogenesis of the fenestra rim as a rule begins within the innermost endosteal bony layer. I did not fail to realize, however, that as far as the end-result was concerned it made no difference whether the periosteal layer when not removed also regenerated or the enchondral layer left intact did not regenerate; neither would it make any difference if both of these bony histologic layers were removed to prevent them from participating in the osteogenetic process, since osteogenesis of the endosteal layer alone within the fenestra rim is sufficient to result in fenestra closure.

In the hope of preventing osteogenesis within the endosteal layer of the fenestra rim I advocated burnishing the fenestra rim with a gold burnishing burr which was proven experimentally in the animal to delay osteogenesis but not to prevent it.[3] As a result of further experimentation with burnishing the fenestra rim with various metals, I found that burnishing the fenestra rim with lead in two Rhesus monkeys prevented osteogenesis[3] and used the lead-burnishing of the human fenestra rim since; however, despite the fact that the percentage of closures was reduced since its use, I never knew which fenestra would remain open and which one would not.

I was never able to prove to myself or anyone else that the fenestra which remained open did so because of having been burnished with lead. I was not unmindful of the fact that many fenestras which I did not burnish with lead remained open as well, nor did I overlook the fact that many otologists who were not using lead were also rewarded with a fair percentage of permanently open windows following this surgery; however, they also were unable to prove either to their own or anybody else's satisfaction why these fenestras remained patent while other fenestras which they made with same technique closed by osteogenesis.

I succeeded in preventing bone regeneration within the fenestra rim by physically impeding osteogenesis with metal obturators; however, I found it technically difficult to make a fenestra which could be snugly fitted with a prefabricated prosthesis.[4] To make a prosthesis to fit individually each newly created fenestra was found not to be practical. I found that I could improve hearing and prevent osteogenesis within the fenestra rim with a properly inserted mobile cartilage larger percentage of postoperative fenestras than heretofore obtainable in narrow fenestras. I could envisage that a wider fenestra may occasionally not complete its mission of fenestra closure after osteogenesis started but could not understand why the creation of a wider fenestra should prevent osteogenesis from starting in its freshly injured fenestra rim.

It, therefore, occurred to me that the same still nonidentified factor which prevented osteogenesis in some of the narrow fenestras was most likely also the one which prevented it in the wider fenestras. Perhaps the reason why more of the wider fenestras stayed open was because a wider fenestra favored this main factor more frequently. I still further reasoned that if there is such a factor, and if a wider fenestra favors the prevention of osteogenesis by this factor, then why did it not prevent it in every one of the fenestras created with the fenestra nov-ovalis technique? This suggested further that perhaps this factor unknown to me did not always exist and in its absence bone regeneration did take place.

To seek an answer to these questions, I had to resort to studying the permanently patent fenestra following the fenestra nov-ovalis technique and also following my first described original technique to see if the same factor prevails in both techniques. I thought that by studying the fenestra gap and rim of the permanently patent fenestra I might be able to recognize and observe some factor still unknown to me which might be held responsible for the prevention of osteogenesis in the fenestra rim. I also hoped that the findings in the fenestra gap of the patent fenestra may perhaps also lead me to the recognition of the factor responsible for the consistent prevention of osteogenesis outside the fistula.

I investigated the fenestra region in 100 ears which failed to show improved hearing following fenestration, in order to observe if possible how osteogenesis was prevented from taking place in some of the fenestra rim margins and why it was not prevented in the others. I wanted to see if there is any difference outside of observable osteogenesis between a closed and an open window.

After studying 100 postoperative fenestras of ears which failed to show improvement in hearing following the fenestra nov-ovalis operation, I found 28 of them closed by bone regeneration and 72 fenestras permanently patent because bone regeneration did not take place In the 28 osteogenetically closed fenestras the findings were the same as in the 517 previously examined closed fenestras. The flap was readily lifted and separated from its attachment to the outer surface of the fenestrated region No bone regeneration has taken place in this region. The fenestra gap was found to be obliterated by newly regenerated bone. The inner surface of the entire flap was found to be evenly smooth as it was when originally placed to cover the fenestra region (see Fig. 1). This held true whether fenestra region was gold-burnished or lead-burnished or not burnished at all.

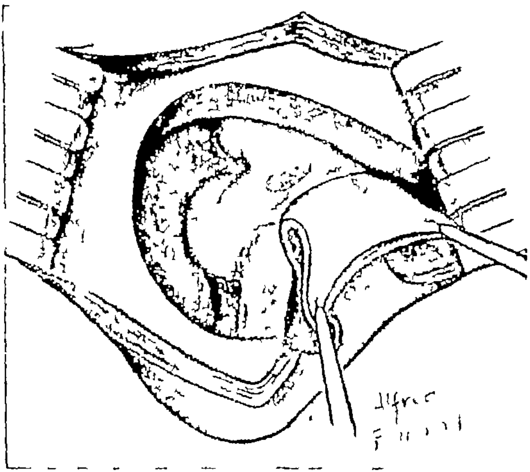

Fig 1. Smooth inner surface of tympanomeatal flap as seen during revision of every osteogenetically closed fenestra.

In every one of the 72 fenestras which remained patent because osteogenesis did not take place, either extramarginal to the fenestra rim or intramarginally in the rim of the fenestra gap, the following significant differentiating observations were made: The tympanomeatal flap was found to be not only adherent to the bony labyrinthine capsule extramarginal to the fenestra gap, but the inner surface of the portion of the flap facing the fenestra gap was found to be invaginated into the fenestra gap and adherent intramarginally to the bony fenestra rim. After slowly and gently separating the flap from its adhesion intramarginally to the bony fenestra rim and lifting it from the entire outer surface of the bony capsule extramarginal to the fenestra gap, a moulded impression of the fenestra gap was observed to exist on the inner surface of the tympanomeatal flap in every one of the 72 patients in whom the fenestra remained patent (see Fig. 2). The membranous labyrinth which was apparently not

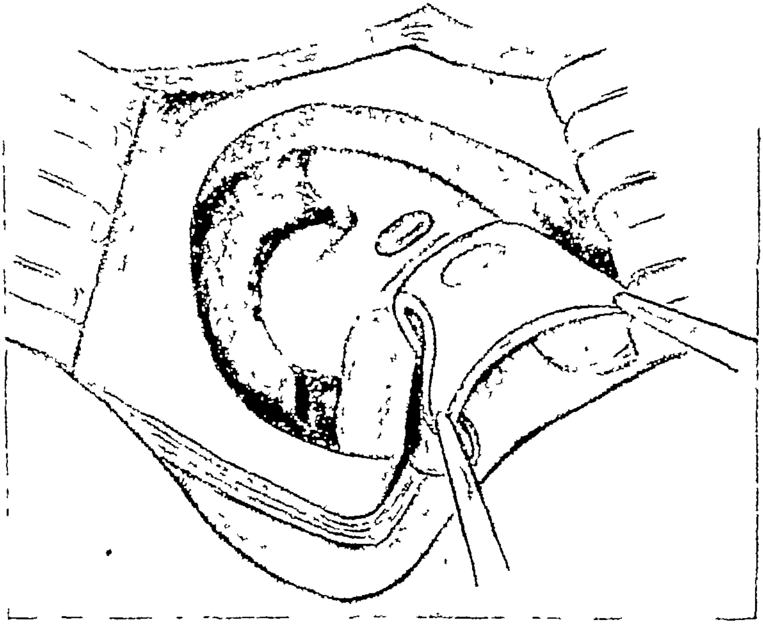

Fig. 2 Moulded impression of fenestra gap on inner surface of tympanomeatal flap as seen during revision of every permanently patent fenestra.

adherent to the flap remained undisturbed in its position within the perilymph space In other words, whenever invagination of the flap into the fenestra gap and intramarginal adhesion of its inner surface to the bony fenestra rim was observed to have taken place, osteogenesis within the bony

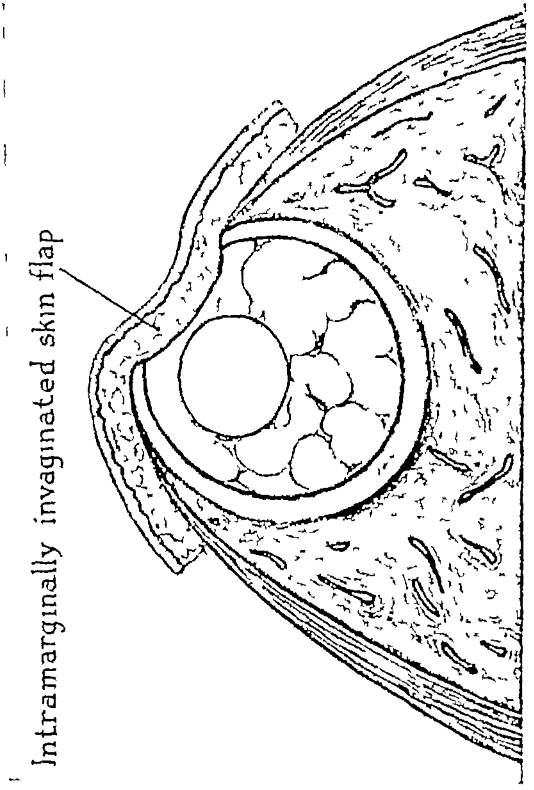

Fig 3 Diagram Showing Cross Section of Permanently Patent Fenestra.
Whenever the inner surface of the tympanomeatal flap became invaginated into the fenestra gap and *became adherent to the lateral walls* of the fenestra rim osteogenesis was prevented intramarginally within the fenestra rim and fenestra remained permanently patent.

fenestra rim was prevented and permanent patency of the fenestra resulted (see Fig. 3). On the other hand, whenever the flap was observed to have become adherent outside the fistula but failed to have become invaginated into the fenestra gap and adherent to the fenestra rim, osteogenesis was pre-

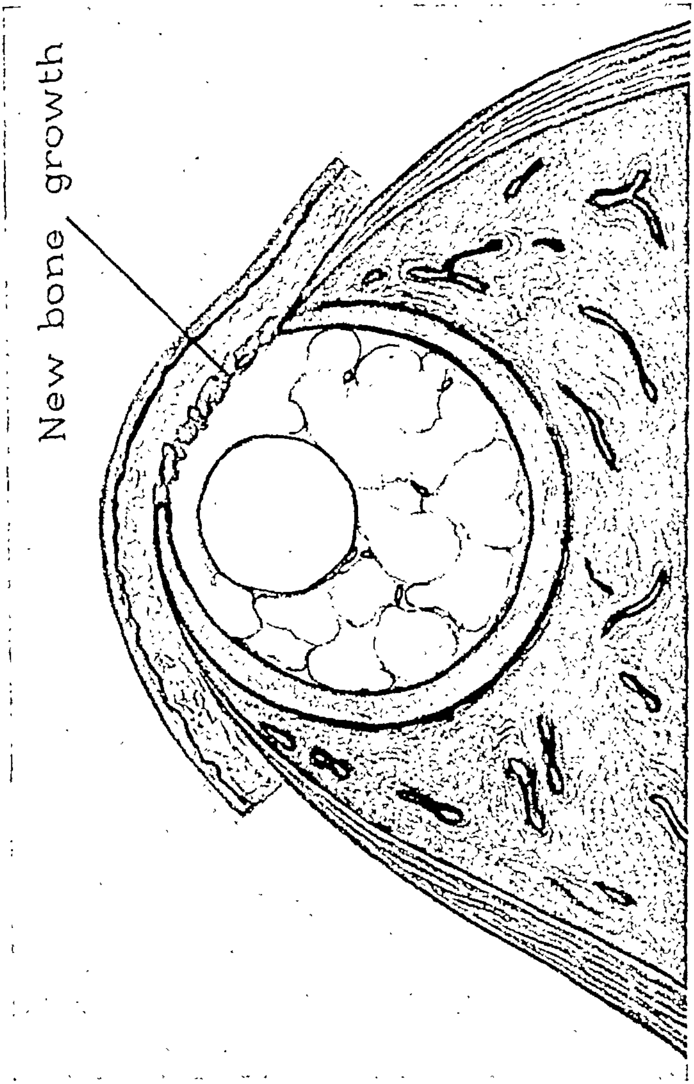

Fig. 4. Diagram Showing Cross-Section of Osteogenetically Closed Fenestra. Whenever the inner surface of the tympanomeatal flap did not become invaginated into the fenestra gap and it did not become adherent to the lateral walls of the fenestra rim, osteogenesis started intramarginally within the fenestra rim and eventually closed the fenestra gap.

vented only outside the fistula and not in the fenestra rim (see Fig. 4).

Since my May, 1950, report before the American Otological Society[8] I have revised five fenestras in patients who enjoyed practical hearing following fenestration for periods varying from three to seven years, but suddenly lost their hearing improvement and became worse without ever regaining their hearing. The fenestra was found to be completely patent and free from osteogenesis in all five cases. The observations made on the operating table were exactly the same as those made in the other 72 permanently patent fenestras.

The above mentioned observations led me to the conclusion that no matter what I, heretofore, did or did not do to prevent osteogenesis from starting in the fenestra rim and eventually closing the fenestra gap following the fenestration operation, unless invagination of the flap accidentally took place and its inner surface became adherent to the fenestra rim within the fenestra gap, osteogenesis took place and closed the fenestra gap.

The ultimate fate of the newly created fenestra was nonpredictable because osteogenesis evidently was prevented only when the pressure exerted by the mastoid wound packing accidentally happened to be sufficient to result in invagination and adhesion of the flap to the fenestra rim.

As a result of these findings it became obvious that: *1.* the bony fenestra edge or rim can heal either by osteogenesis which obliterates the fenestra or by invagination of the inner surface of the flap into the fenestra gap and its adhesion to the bony edge of fenestra rim, which prevents osteogenesis and permits fenestra to remain patent; *2.* more windows stayed permanently open with the use of the fenestra novovalis technique because the much wider fenestra created with this technique favored accidental invagination of the flap into the fenestra and its adhesion to the fenestra rim much more frequently than the previously made much narrower fenestra; *3.* osteogenesis as a rule did not take place extramarginal to fenestra rim because adhesion of the flap to the bony labyrinthine capsule outside the fistula prevented it.

union as a result of postoperative infection it often became invaginated and adherent to the bony edges of the mastoid cortex and a permanent postauricular fistula resulted (see Fig. 5).

How can we now, with this new knowledge gained about the prevention of osteogenesis, consistently and deliberately create a permanently patent fenestra?

1. Make fenestra on surgical dome of vestibule.

2. Make it as wide as possible. In order to do so, you must remove the anterolateral wall of the ampullated end of the bony external semicircular canal, thus exposing a wider area of the perilymph space anterior to the normally placed membranous labyrinth. Fenestra should be about 2 mm. wide and about 6 mm. long.

3. Place tympanomeatal flap so that it covers and contacts the entire bony region outside the fenestra gap.

4. Invaginate that portion of the tympanomeatal flap which faces the fenestra gap, sufficiently only to contact intramarginally the fenestra rim (see Fig. 6).

5. Place a cotton inlay moistened in Ringer's solution over the invaginated portion of the tympanomeatal flap and mould it so that it forces the inner surface to contact and stay in contact with the fenestra rim edges intramarginally. This favors its adhesion intramarginally to the bony fenestra rim (see Fig. 6).

6. Secure and hold cotton inlay in its position with a piece of paraffin mesh gauze moulded over it.

7. Pack rest of cavity with paraffin mesh gauze to hold the initial packing in its position.

8. Remove all packing except the first piece on sixth or seventh day.

9. Remove last piece on the tenth to twelfth day.

CONCLUSION.

I have finally observed and learned that invagination of the tympanomeatal flap into the fenestra gap and intramarginal adhesion of its inner surface to the relatively avascular bony fenestra rim prevents osteogenetic closure of the newly created fenestra. As a result of this observation it also became obvious that the reason osteogenesis did not as a rule take place in the relatively avascular bony labyrinthine capsule outside the fistula following fenestration surgery even when osteogenesis took place within fenestra rim was because the flap which became adherent to it prevented it. With this knowledge at hand we should now be able deliberately to prevent postoperative osteogenetic repair of the fenestra novovalis and as a result obtain a much larger percentage of permanently patent fenestras than ever before.

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HISTOPLASMOSIS IN THE ADULT: CASE REPORT AND REVIEW OF THE LITERATURE.*†

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Histoplasmosis has been defined by Worgan[1] as a fungus disease involving the reticuloendothelial systems. It is of particular interest to otolaryngologists because of the ulcerative lesions of the mucous membranes of the upper respiratory passage and alimentary canal. Frequently the cervical vertebrae, frontal and maxillary bones and the middle ear tract have been affected. We will summarize briefly the findings of various observers and then report one of the four cases which have been recognized at the University Hospital during the last two years.

Histoplasmosis was discovered by Darling,[2] in 1905, while looking for evidence of kala-azar in smears of the spleen, liver and bone marrow. In 1906, he reported finding enormous numbers of small bodies, generally oval or round, most of which were within the alveolar epithelial cells; however, some appeared to be free in the plasma of the spleen and rib marrow. Darling thought the disease was caused by protozoan parasites. Twenty years elapsed before the next case was reported by Watson and Riley. According to Worgan,[1] DeMonbreun proved that the causative agent was not a protozoan parasite but a fungus which was called histoplasmosis capsulatum. It existed in two forms: one, a yeast-like form which grows in the reticuloendothelial system of man, and the other, mycelial, growing outside of the body.

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Worgan[1] reported that DeMonbreun, in 1938, isolated both the yeast-like and mycelial forms of the disease. He obtained a pure culture of the fungus which grew like "small white cotton tufts," distributed over the surface of dextrose and Sabouraud's agar. Although there were numerous colonies, on inoculation the mycelium has no aerial hyphae. He was unable to find the yeast-like forms in the original culture but later did find them. He described the organisms as oval bodies measuring about 3 microns in diameter, the cell being surrounded by a thick membrane containing one or more fat droplets and usually a *single protoplasmic granule.* Supravital staining with methylene blue revealed deeply stained granules. Each cell contained one or more vacuoles which took up the stain. If the yeast-like forms were allowed to lose their moisture, the mycelial form soon made its appearance.

The portal of entry of the fungus is not known. Some authors, workers, persons and students think that the mouth is probably the most frequent site of entrance because of the ulcerations of the mucous membrane of the mouth, pharynx and gastrointestinal tract. Others feel that the respiratory tract is the pathway of entrance because the pharynx, larynx and lungs often have lesions.

Hodgson, Weed and Claggett[3] made a survey of the cases reported up to 1949 and found 123 cases. They learned that the disease was rather widespread. Cases were reported from 25 states and 17 foreign countries; three-fourths were from the United States. Leopold[4] reported that in studies made by Gass (1943) and Lumsden (1939) a much higher figure of pulmonary calcification was found in nonreactors to tuberculin residing in the area of the eastern slope of the Mississippi River basin and the adjoining states and the western slope south of Iowa than in the rest of the country. The ages of the patients varied between seven and one-half weeks to 77 years, and about one-sixth were less than one year of age.

The disease appears to be more prevalent in males than females. Parson and Zarafonetis[5] reported histoplasmosis

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Grossly the typical pulmonary lesions have a soft yellowish white foci which resemble tubercules.[1] The average lesion measures from 5 to 15 mm. and is surrounded by a translucent fibrous capsule. Worgan[1] at autopsy found the parasite to be a pear-shaped crescentic organism, two to four microns in size and surrounded by a thick refractile capsule. They all contained one or more nonstaining vacuoles and one or more dark chromatin masses. Usually there was an invasion of the tissues by mononuclear cells which contained many of the parasites and red blood cells.

DISTRIBUTION OF PATHOLOGIC LESIONS.

Dean[6] reported a case with involvement of the larynx in which the epiglottis was thick, nodular and pale, and the whole interior of the larynx appeared to be white and nodular. He also stated that the false and true cords were so extensively involved that they appeared to be entirely obliterated. The glottic chink was converted into an irregularly rounded tube. The organism was recovered from the granulations about the tracheotomy wound.

Duncan[7] also described a case reported by Miller, Johnstone and Bostick, in which there was an ulcerating nodule about 1.5 cm. in diameter on the dorsal aspect of the tongue. The diagnosis was made by scraping the ulcer and isolating the fungus. There was marked lymphadenopathy, and enlargement of the liver but not the spleen.

Parson and Zarafonetis[5] made a splendid report of the distribution of the lesions in their cases. A summary of their report was as follows: Of 61 cases, 19 had some skin lesions present; nine had ulcers; four had papular eruptions; six had purpura occurring in the last two or three days of life. Petechial hemorrhages and bullous lesions were found once. The ulcers frequently were deep with sharp, steep edges, having a punched out appearance. In one case of involvement of the tongue there was considerable edema around the border of the lesion. They also reported involvement of the middle and external ear canal in eight cases and a true otitis media in five cases. Organisms were found in the pus in one case of middle ear infection. Ulcers of the mucous membrane of the mouth most frequently involving the tongue were found in 14 cases; four other cases had ulcers of the mucous membrane of the nose and mouth. Perforation of the nasal septum was noted in three of their cases. The perforations of two were known to have been produced by infection with histoplasmosis capsulatum. There were granulomatous and ulcerative lesions of the pharynx and larynx in 10 cases. Superficial lymph nodes were discovered in 29 of the 61 cases with enlargement in 16 cases. Cervical nodes alone were affected in five cases, and in conjunction with other regional nodes in four other instances.

Agress and Gray[8] reported histoplasmosis in a boy seven months of age with a saddleback deformity of the nose, perforation of the septum, mucopurulent rhinitis, ulcers of the buccal mucosa and lesions about the mouth suggestive of rhagades. The liver and spleen were enlarged, but there was no lymphadenopathy. The Kahn and Wassermann tests were negative.

METHOD OF DIAGNOSIS.

The diagnosis of histoplasmosis can be established only when the clinician suspects it and includes it as a possibility even in the absence of skin and mucous membrane lesions. Diagnosis by biopsy has been the most successful of all methods during life. Sternal marrow aspiration often enables the examiner to identify the organisms within phagocytic cells of reticuloendothelial origin, and culture of this aspirated material makes this an even more reliable procedure. Blood smears may prove a source of recognizable histoplasma capsulatum in the generalized type but thick film preparations increase the probability of success. Blood cultures likewise help. In the same regard, culturing of biopsy material obtained from ulcerated involved areas may aid in establishing the diagnosis. If a recent or old infection is suspected, one may use an intradermal injection from a filtrate of cultured medium in which the fungus has grown. A positive test is indicated by erythema and a wheal at the site of the injection.

TREATMENT.

Treatment with pentnucleotide, fuadin, irradiation, neostam, sulfonamides, sodium thiosulfate, iodides, ionized metals, potassium tartrate, neoarsphenamine, acetarsone, autogenous vaccines, blood transfusions and various other drugs bears witness to the lack of a specific cure. Histoplasmosis is almost universally fatal, usually within a few months and rarely some years after the onset of the disease. Parson and Zarafonetis[5] reported that in most cases clinical signs of infection lasted less than one year, the average in 39 cases being about five months. In one group, however, there were four cases that lasted four, eight, 10 and 16 years, respectively.

CASE REPORT.

Case No. 50-8604: A 34-year-old farmer was well until Sept. 17, 1949, when he developed a dry cough, chills, sweats, weakness, and a temperature of 103.01°. He was unable to work. In October, 1949, a palpable spleen and temperature of 106° were noted.

A sore throat first occurred in January, 1950, when biopsy of a laryngeal lesion revealed no malignancy. In March, he consulted a neighboring clinic. A biopsy of the larynx was obtained and the tissue section demonstrated histoplasma capsulatum. Within a few days after returning home, he was unable to talk and had progressive difficulty in swallowing food. He complained of a sore throat with pain radiating to both ears. He was unable to swallow even saliva. Diplopia occurred on July 7, 1950; and 10 days later there was sudden loss of function of the right arm and leg without unconsciousness. A few days later dyspnea began with rapid aspirations. He had a chill and became disoriented. He had received alone or in combination numerous antibiotic and sulfonamide drugs with progression downward. On admission, there was emaciation, dehydration, mental confusion, aphonia, fever and rapid shallow respiration. Hypertension of 170/100 was present and continued throughout the illness. Right external rectus muscular-paralysis and a droop of the right face excluding the forehead were evident. Two eroding ulcerative lesions were on the tongue. A well healed thyroidectomy scar was noted. The lungs contained many moist rales especially on the right. Complete flaccid paralysis of the right arm and right leg were present.

The appearance of the larynx in our case was very similar to the one reported by Dean.[6] It was generally pale and edematous, and one could not distinguish between the false and true cords, the normal landmarks being obscured. The larynx was tubular in appearance with some obstruction to the airway. The arytenoids appeared hypertrophic and edematous. There was an irregular, cauliflower, pale mass about 1.5 by 1 cm. in size involving the intra-arytenoid space, and extended into the chink of the glottis. The patient was having some difficulty in swallowing and an esophagoscopy was attempted, but we were unable to pass the scope because of a definite stricture at the level of the cricopharyngeus. The mucous membrane was pale and edematous, and it was impossible to pass a 3 mm. feeding tube.

Gastrostomy was performed and in a few days all necessary fluids plus a more than adequate diet were being given.

There was a hypochromic anemia, uremia, hypoproteinemia, albuminuria, and continuing temperature elevation with no marked change from the previously described critical picture. Bronchoscopy was necessary on several occasions, as well as almost constant pharyngeal suction.

Direct examination and culture of the blood and bone marrow failed to reveal any organisms. Fungus was cultured from the tissue biopsy on several occasions but at no time would it produce tuberculated chlamydospores and, therefore, could not be positively identified as histoplasma capsulatum. A tissue section taken from the larynx in March, 1950, and sent to us by Dr. L. B. Woolner was positive for histoplasma capsulatum.

In addition to the usual supportive and other therapeutic measures, we attempted the administration of ethyl vanillate, a fungicidal agent which shows considerable promise in generalized histoplasmosis infection in children.[9] We were unable to obtain satisfactory blood levels, however, and death occurred seven weeks after admission. Autopsy permission was refused.

We have attempted to call attention to this infection whether generalized or localized. We feel that it is of special interest to the otolaryngologist in view of the fact that many of the lesions involve the mucous membrane of the upper respiratory and digestive tracts, sinuses, external ear and middle ear.

We thank Dr. L. B. Woolner, of the Mayo Clinic, for the use of the tissue section, and Dr. A. P. McKee, of the Department of Bacteriology, College of Medicine, State University of Iowa, for his work in culturing and identifying the fungus.

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BRONCHIAL ADENOMA.*

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Bronchial adenoma is one of the most important and most interesting tumors found in the bronchi. The histopathologic diagnosis is difficult, and these lesions are often mistaken for carcinoma. It is important to discover them early, because if not treated they produce bronchial obstruction of marked degree, and subsequently bronchiectasis, abscess and empyema; furthermore, they *must be* differentiated from carcinoma because they require less radical treatment.

The rôle of bronchoscopy is especially important in the diagnosis and treatment of these tumors because they almost always arise in the larger bronchi. Certainly everyone admits that they can generally be seen and biopsied by bronchoscopy, regardless of the efficacy of bronchoscopy in treatment.

Our experience with these tumors has been presented in previous articles: Jackson and Konzelmann,[1,2] Jackson[3,4] (in German and Italian), Jackson, Konzelmann and Norris,[5] and Jackson and Norris.[6] In the last of these previous papers, given before the annual meeting of the American College of Chest Physicians in San Francisco, Calif., last June, our previous papers were summarized, and 40 cases were reported. Seventeen of those cases had never been reported before, and the remaining cases those previously reported, and presented again with late follow-up.

Symptomatology and Diagnosis. The symptomatology of bronchial adenoma has already been extensively discussed,

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and it is well known that this type of tumor produces *hemoptysis* early, is generally accompanied by *cough*, more often productive than not, and above all by the physical and X-ray signs of *bronchial obstruction*. The *diagnosis* is made on the basis of these findings, confirmed by the bronchoscopic appearances, and the opinion of the pathologist based upon study of specimens removed for biopsy. Cytologic studies of secretions have not been so helpful as in carcinoma, but by the nature of the lesion, actual tissue specimens can generally be obtained, because the tumors generally arise in the accessible portions of the bronchial tree.

Pathology. It has always seemed to us that the crux of the confusion concerning these tumors lay in the histopathology. The typical histopathologic picture has been described by Konzelmann.[1] The subdivision into different types, as for example "*carcinoid*" and "*cylindroma*" types as suggested by Hamperl[7] is useful. There has been much discussion as to whether bronchial adenomas should be considered benign or malignant, but we believe that they should be considered "borderline" tumors[8] for the present. Malignancy ordinarily depends chiefly on tendency to local invasion, and ability to metastasize. Certainly the appearance of local invasion is given by these tumors, as was noted in the early study of the subject by Wessler and Rabin.[9] These authors pointed out, in 1932, that it was characteristic of bronchial adenoma to extend between the bronchial cartilages and for some of the tumor to be extrabronchial; however, we believe that this in itself cannot be considered a malignant characteristic, because it is due simply to the fact that the tumor arises from structures which occur between the cartilages rather than on the mucosal surface. Neither can involvement of contiguous lymph nodes be considered final proof of malignant character. Many of the cases of distant metastasis, as for example to the liver, have been proven to be cases of erroneous diagnosis, but it may be true that a few of the cases of liver metastasis which have been reported have represented true metastasis of a bronchial adenoma. We admit this, though such metastasis has not been noted in any of our own cases.

Treatment. We believe that irradiation has been of no proven value; that in many cases bronchoscopic removal is curative, and that in many other cases it constitutes efficacious preoperative or palliative treatment; that bronchotomy as shown by Goldman[10] and some others offers a good prospect of cure by a conservative surgical procedure in suitable cases; and that surgical resection by lobectomy or pneumonectomy is definitely indicated in many other cases, because of impossibility of complete local removal, compression due to persisting extrabronchial tumor; or irreversible changes in the distal portion of the lung. Finally, we very definitely believe that in dealing with bronchial adenoma, cases should be individualized in selection of treatment method, and resection should not be resorted to automatically as in carcinoma.

Clinical Experience. As mentioned above, our series of reported cases now includes 40 patients; 36 of these are considered to be of the carcinoid type, and four of the cylindroma type. Of these 40 cases, 26 patients were treated by bronchoscopic measures only, eight by lobectomy (with one postoperative death), and three by pneumonectomy (with one postoperative death); two patients had biopsy only and were lost track of. Of the five patients in the entire series of 40 who have died, two died postoperatively, though neither of the postoperative deaths occurred at the Temple University Hospital. All nine of the patients operated upon by Dr. W. Emory Barnett and Dr. George Rosemond at the Temple University Hospital (seven lobectomies and two pneumonectomies) survived. Two of the patients who died of disease were typical cylindromas and were treated bronchoscopically because the tumors were too close to the carina to permit pneumonectomy, though they would have been subjected to pneumonectomy if this had not been the case. The other two patients with cylindroma are living and symptom-free, apparently quite well under control by bronchoscopic treatment at long intervals.

Summing up, of the 40 patients in the series, five have died, two of them postoperatively; two were lost track of, and 33 are living and well, though some require occasional treatment and all return for occasional check-up examination.

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ANTRO-ALVEOLAR FISTULAE.*

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While the majority of antro-alveolar fistulae follow dental extractions, they may result from infection, cysts, or tumors of the upper jaw, trauma, or as a complication of operations on the antrum. Successful management depends upon careful study of the individual case and the application of a procedure calculated best to meet the existing conditions. This may vary from simple approximation of tissue to the use of fairly extensive plastic methods. Control of antral infection is essential. Treatment must be based upon sound anatomic and physiologic principles.

The importance of cooperation between the dentist, the oral surgeon, and the rhinolaryngologist cannot be overemphasized, particularly in the field of prevention.[1-3] The potential dangers of inadvertently entering the antrum in the extraction of teeth in close relation to the floor, and of subsequent sinus infection should be kept in mind. Should extraction of such a tooth be indicated, the dentist could well assure himself that antral infection was not already present. In the case of already existing sinusitis, measures to control this, such as intranasal antrostomy prior to extraction, would avoid many of these complications. The dentist and the oral surgeon should realize that infection of the antrum is a rhinological problem and any involvement should be managed by one trained in rhinology.

In large antra with thin walls the roots of the bicuspid

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teeth and the first, second, and sometimes the third molars, or even the canines, may mound up in the floor of the sinus. Sometimes the bone may be missing with only the mucoperiosteum separating the roots from the antrum.

Fistulae of dental origin may occur as a result of rough attempts at extraction, the breaking off of roots and pushing them upward into the antrum, too strenuous curettage of sockets, interlocking roots sometimes looped over a thin antral floor, or roots with marked hypercementosis. Fusion of molars and bicuspids may be a cause of fistula following extractions. In a case of the author's, the dentist in attempting extraction of the first molar had removed the first, second, and third molars, together with surrounding process, all in one

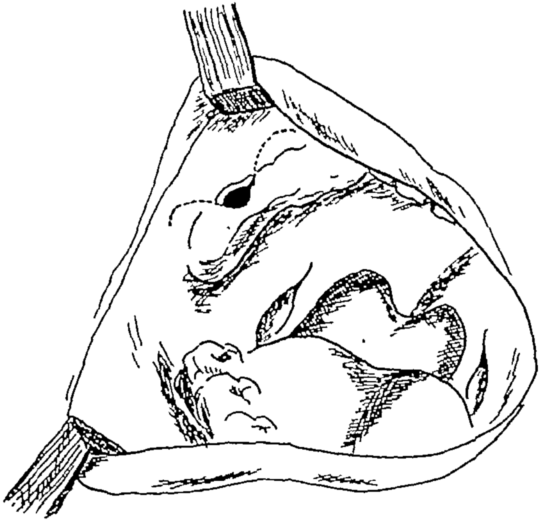

Fig. 1. Showing modified canine incision extending downward to include outer margin of fistula.

mass. Most of these fistulae could be prevented by a more careful technique or previous Roentgen study on the part of the exodontist. Approaching a retained root from the side, by removal of some of the lateral plate, and manipulating it downward from above, generally will avoid pushing the root into the antrum. Any granulomatous tissue in the socket should be carefully removed, avoiding the forcible use of sharp curettes which might result in pushing through the thin floor into the antrum. Study of the dental Roentgenograms should allow the exodontist to plan his procedure so as to avoid undue trauma to the antral floor. Sometimes splitting the crown of a molar will allow removal in the plane of the long axis of distorted roots.

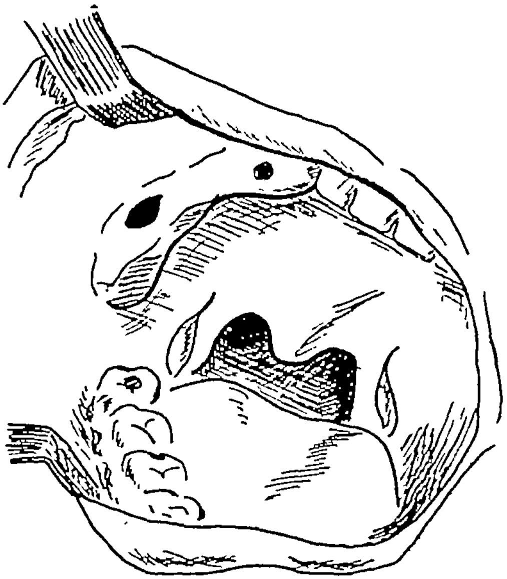

Fig. 2. Soft parts elevated exposing fistula. This drawing was made from a case with multiple fistulae. One in the site of the second molar and a smaller one in the socket of the canine.

When a root or, as sometimes occurs, an entire tooth has been forced into the antrum, it is preferable to remove this by opening the antrum through the canine fossa, although occasionally it is possible to effect removal through an intranasal antrostomy. Efforts to manipulate any foreign body down through the fistula usually adds to the trauma and makes healing more difficult.

While rarely seen, to be sure, the possibility of neoplastic changes occurring in an antro-alveolar fistula should be kept in mind.

One patient in our series,[4] a man of 41, was seen because of pain and swelling in the upper jaw and neck of one month's duration. He had had a discharging antro-alveolar fistula following the extraction of teeth 20 years before. Examination revealed a large ulcerative tumor mass in the alveolar process with a fistulous tract extending into the antrum. The submaxillary area was greatly swollen with a mass of

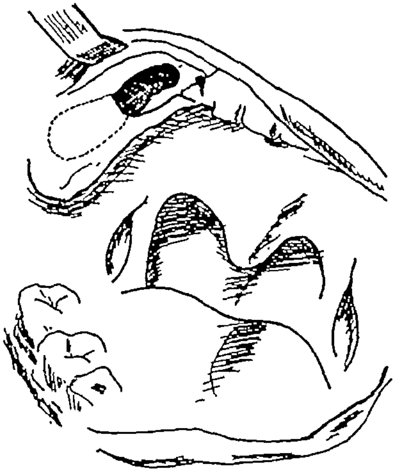

Fig. 3. Necrotic bone surrounding fistula removed exposing interior of antrum. Reversed or hinged flap from posterior tuberosity outlined.

broken-down metastatic nodes. Biopsy was reported as epidermoid carcinoma. Extensive electrocoagulation of the primary lesion, followed by irradiation, using radium as well as external Roentgen therapy resulted in temporary improvement; but attempts to eradicate the metastatic mass in the neck were unsuccessful, and the patient succumbed in eight months.

Many fistulae may heal spontaneously or with a modicum of intelligent treatment directed to the antrum, but a tract of any size, from the mouth with its rich and variegated bacterial flora to the antrum, is most liable to result in infection of the sinus, if indeed this is not already present, due to chronic sinusitis.

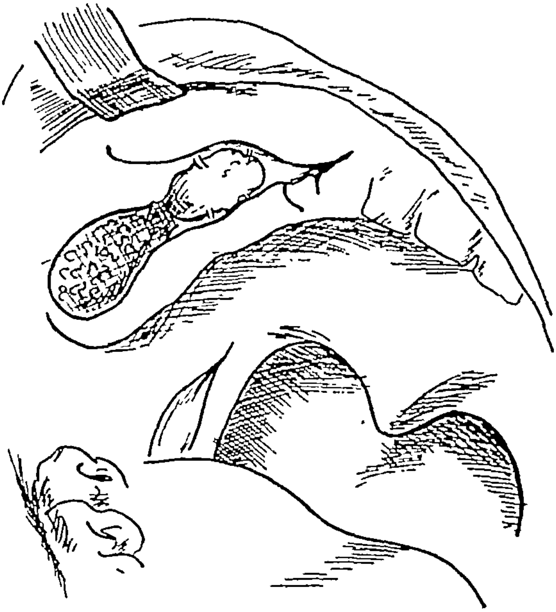

Fig. 4. Hinged flap turned forward covering fistula and presenting raw surface for sliding buccal-gingival and palatal flaps. Anchored with one or more catgut sutures.

The advantages of early attention in these cases are obvious. This may result in spontaneous healing of the fistula or a satisfactory response to simple methods of closure. Persistence of the condition not only increases the technical difficulties in achieving a closure but also exposes the patient to all of the annoyances and hazards of a chronic sinusitis.

Roentgenograms of the sinuses are mandatory. If there is little evidence of a pre-existing chronic sinusitis conservative measures may be employed. These include irrigation of the antrum, parenteral antibiotic therapy based upon culture of the organism, as well as intranasal use of vasoconstrictor sprays. Packing, irrigation, or any manipulation of the fistulous tract, including the application of caustics, is contraindicated; not only are these useless but oftentimes may add to the difficulty.

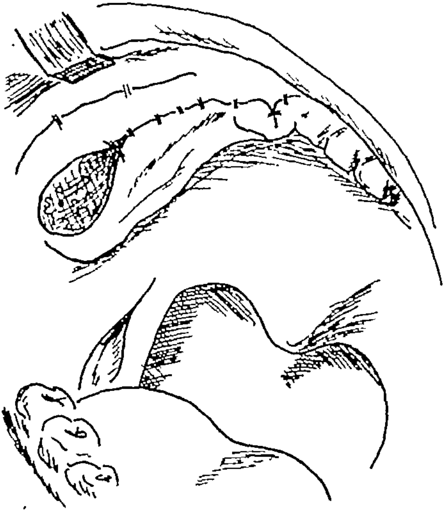

Fig. 3. Bipedicled buccal-gingival and palatal flaps brought together over raw surface of hinged flap and sutured with silk. Mucous membrane will grow across denuded area of posterior tuberosity.

When the antral lining is markedly thickened, and there is evidence of pre-existing chronic sinusitis, an intranasal antrostomy is indicated. Little is to be gained by procrastination. Intranasal drainage should be established as early as possible. The removal of secretions by the suction cannula through the antrostomy opening is, in my opinion, preferable to lavage, although many operators employ irrigation. Some fistulae, if small and seen early, may close spontaneously following such therapy. Usually, however, closure will require the elevation of adjacent mucoperiosteal flaps, with sufficient lowering of the alveolar process to permit approximation without tension.

A number of techniques, such as the Dunning[1] and the Welty,[5] have been employed, based on the principle of covering the defect with adjacent mucosa from the palate. The use of the saddle-plate, as described by Dunning, to protect the suture line, is helpful when there are adjacent teeth for its attachment. Ashley[6] described a procedure in which a long mucoperiosteal flap was taken from the palate and the anterior portion, denuded of mucosa, used to plug the opening. Isografts of cartilage inserted in the tract have been used to close the fistula. McQuiston[7] employs sliding flaps from the palate and the buccal surface of the alveolus, about 1.5 cm. in width, the buccal incision being similar to the usual one employed in the Caldwell-Luc operation. After curettage of the fistulous tract and removal of infected granulatious tissue, these flaps are approximated with silk mattress sutures.

Local anesthesia is very satisfactory in all of these procedures. Good oral hygiene is essential. Any dental caries should be eliminated before attempting any plastic procedure on the alveolar process.

The fistula of long duration will have developed a tract lined with mucous membrane which will require removal. The antrum is always infected and usually should be approached through the canine fossa using the Caldwell-Luc technique. Occasionally one may encounter a small fistula of this chronic type, in which the "folding-door" type of incision,[8] followed by undermining the lining of the tract with a narrow cataract knife and turning this cored tract inward, may prove successful. The flaps of the "folding-door" are then closed by mattress sutures of deknatel. A slight amount of the alveolar process may be removed with ronguers to facilitate approximation. Sutures are removed in four or five days.

In all of these procedures patients are cautioned against sucking on the operated area, manipulating it with the tongue, or forcible blowing of the nose.

The management of a large fistula of long duration may prove rather complicated and resist simpler methods of closure. In dealing with this type the Caldwell-Luc operation° should be combined with the plastic procedure on the fistula. Formerly it was my custom to do this in two stages, the usual Caldwell-Luc operation being followed in a week or 10 days by the plastic procedure on the fistula. This had the disadvantage of making impossible a strong buccogingival flap, this being limited by the horizontal canine fossa incision. For some years I have employed the one-stage operation, modified as follows for these large fistulae.

The indicated antibiotic therapy is given preoperatively and carried on postoperatively.

Local anesthesia is employed, after preliminary medication with nembutal (pentobarbital sodium), gr. 1ss, one and two hours before operation. The lateral wall of the inferior meatus of the nose is anesthetized by applying a thin strip of cotton moistened with cocaine 10 per cent and adrenlin chloride 1-1000. Two to three cc. of procaine 2 per cent are then injected slowly into the infraorbital foramen. This may be done directly through the cheek after the foramen is located by palpation. The posterior palatine foramen is then injected, followed by infiltration in the anterior part of the hard palate and along the buccogingival groove.

A modification of the usual canine fossa incision is employed, curving the mesial portion downward to include the lateral margin of the fistula. This allows for a more substantial buccal flap than is possible with the usual horizontal incision and also avoids the scar tissue inevitable in the two-stage procedure. A sharp submucous dissector is used to elevate the periosteum until the wall of the antrum is sufficiently exposed. The defect in the bone is generally lateral, as most teeth are extracted laterally. This allows adequate exposure of that area, so that any necrotic bone may be removed with ronguers. Usually this gives an opening adequate to remove the infected antral lining and make the intranasal window.

After the antral part of the operation is completed a hinged or reverse flap of mucous membrane is dissected from the alveolar process, usually posterior to the fistula, and turned forward to close the weak posterior angle and provide a raw surface for sliding flaps of buccogingival and palatal mucous membrane. The apex of this flap is anchored to the lower or unelevated margin of the original incision just anterior to the fistula with a No. 0 catgut submucous suture. An incision is now made in the buccal mucous membrane, somewhat parallel to the original incision, forming a ribbon, or sliding flap, approximately 2 cm. in width, by dissecting with scissors submucously and leaving both ends intact. This buccogingival flap should be fairly generous, care being exercised to avoid Stenson's duct. With this bipedicled flap, with both ends intact and only its midportion sliding downward, adequate blood supply is possible. It has proven quite as satisfactory as the delayed flap.

The palatal flap, also bipedicled, is cut about midway between the midline of the palate and the alveolar process and stops short of, or swings lateral to, the posterior palatine foramen. Both flaps are so freed that their central parts may be approximated without tension over the hinged or reverse flap, using single or mattress deknatel sutures. If projecting process prevents this it may be lowered. Usually in the large fistula only the palatal plate of the process will require trimming.

The buccal incision may be loosely approximated with one or two sutures. The tissue in this region is so free and pliable that it heals readily. It is advisable, however, to caution the patient to avoid, as much as possible, motion of that side of his face, or pushing the tongue towards the cheek. The palatal incision is not sutured and usually heals readily and with much less discomfort for the patients than with the median palatal incision.

This procedure allows for adequate exposure of all diseased areas and provides a bed for the mucous membrane flaps, bringing raw surface against raw surface. It seems to meet the requirements of plastic surgery and, in our hands, has proven most satisfactory. It does not require a large palatal flap and can be used even in cases having a torus palatinus.

In the management of any antro-alveolar fistula of long duration it is essential to eradicate thoroughly any osteomyelitis. Sometimes this may require considerable removal of bone of the anterior antral wall. In the past when employing the two-stage operation, this procedure, at times, has resulted in an operative defect in the canine fossa, necessitating repair with a delayed flap.[10] In our experience this has not occurred since adopting the one-stage procedure as above outlined.

In endentulous patients it is important to preserve sufficient alveolar process to insure the satisfactory use of a denture. The tuberosity of the maxilla may be considered as the posterior buttress, very necessary for an upper denture. If this is preserved a denture usually can be made to function satisfactorily. In this connection it is well to remember that all patients may be candidates for full dentures at some later time.

AFTER-TREATMENT.

Patients are allowed out of bed the day following operation. Soft diet is given, but they are instructed to chew on the unoperated side. Frequent mouth washes are prescribed, and any accumulated food is gently removed with cotton swabs. The incisions may be painted with methylene blue or gentian violet 1 per cent, once or twice daily. Sutures are removed on the fourth or fifth day. Any accumulated blood or debris is removed from the antrum by cannula suction intranasally twice a day. Healing usually takes place by the seventh or eighth day, and if dentures are worn they may then be resumed.

In fistulae resulting from cysts or tumors the same general principles apply, although first consideration must be given to thorough eradication. This may result in very large defects either in the canine fossa or in the palate, so that the use of a prosthesis may be required. In cases of malignancy it is advisable to maintain adequate openings for the purpose of postoperative observation. In such cases flushing the cavity once or twice daily with potassium permanganate (1-10,000), or at times the insufflation of sulfanilamide powder, is helpful in overcoming any odor and keeping the cavity clean.

CONCLUSION.

The majority of antro-alveolar fistula are of dental origin and are preventable.

Successful management depends upon the control of antral infection, whether pre-existing or resulting from the fistula. The greatest hazard is gravity drainage.

The problem of management may be quite simple or extremely complicated. No one procedure is adaptable for all cases. Some may heal spontaneously or respond to conservative measures and simple methods of closure; others may require carefully planned plastic procedures.

A one-stage technique which has proven satisfactory in the most resistant type is presented.

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EMOTIONAL FACTORS IN OTOSCLEROSIS.*

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Having set forth in several publications concepts underlying the effects of emotional disturbances upon the organ of hearing, and especially in respect to tinnitus, vertigo, and certain types of obstructive, neural and sudden deafness, it is now my purpose to call attention again to these in relation to otosclerosis; not only to examine them as concomitants or resultants of this disease, but also as possible factors in its etiology.

Were it not for our abysmal ignorance as to the cause of otosclerosis, and our utter helplessness in curative measures, I should hesitate to present the tenuous data I here set forth; however, even the faintest lead should not be neglected. Time and experimentation will prove whether it will develop into something worthwhile.

It is to be emphasized that up to the present hour otoclerosis has never been shown to result from any specific cause. It may develop, or cease to develop, in the presence or in the absence of other disease entities. It is markedly less prevalent in the Negro race, and less prevalent in males than in females, and as far as we know, in all races. It has never been produced or revealed in animals in a form surely recognizable as the same lesion. It is thought that its etiology may be hidden in some inherited factor.

My own observations lead me to believe that in addition to the psychic reactions observed in chronic deafness from any

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EMOTIONAL FACTORS IN OTOSCLEROSIS.*

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Having set forth in several publications concepts underlying the effects of emotional disturbances upon the organ of hearing, and especially in respect to tinnitus, vertigo, and certain types of obstructive, neural and sudden deafness, it is now my purpose to call attention again to these in relation to otosclerosis; not only to examine them as concomitants or resultants of this disease, but also as possible factors in its etiology.

Were it not for our abysmal ignorance as to the cause of otosclerosis, and our utter helplessness in curative measures, I should hesitate to present the tenuous data I here set forth; however, even the faintest lead should not be neglected. Time and experimentation will prove whether it will develop into something worthwhile.

It is to be emphasized that up to the present hour otoclerosis has never been shown to result from any specific cause. It may develop, or cease to develop, in the presence or in the absence of other disease entities. It is markedly less prevalent in the Negro race, and less prevalent in males than in females, and as far as we know, in all races. It has never been produced or revealed in animals in a form surely recognizable as the same lesion. It is thought that its etiology may be hidden in some inherited factor.

My own observations lead me to believe that in addition to the psychic reactions observed in chronic deafness from any

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or acquired. It is certain that emotional instabilities present before the onset of deafness will influence the reactions to the deafness, of the patient, his family and friends.

It is now believed that there are genetically controlled stimulii and reflexes which lead to basic instincts and behavior patterns. These patterns are in large degree inherited as potentialities. It follows that a child of a parent with a hypersensitive autonomic system will in a considerable number of cases inherit such potentialities or predispositions. In addition he will be subjected environmentally to the exaggerated emotional reactions of the parent. He will react similarly to the parent who has the more to do with his social development (usually the mother). Mothers are commonly more closely associated with their daughters' lives than with their sons'. (Here there are many implications.)

The age incidence of otosclerosis and its varying locations and periods of activity as revealed by clinical experience and in Guild's[3] histologic data indicates that the lesion develops only after birth and in an increasing incidence ratio varying with certain age decades; that the lesion is latent in the bone long before it is recognizable morphologically, and active long before it is recognizable clinically. Almost all of the cases reported as otosclerosis before the age of 10 are open to question, and the ones under eight are all very questionable. Also it suggests that if autonomic neural factors are in fact implicated in the etiology or activity of the lesions, the age incidences, distributions and periods of activity would be variously spaced just about as they are. No other etiologic factors so far suggested fit into the pattern as well as this.

Long before birth, although the neurovascular like the respiratory systems are present and ready to function, they are primarily completely under maternal influence until after birth. Immediately after birth and all during infancy and childhood the emotional factors develop with increasing powers to cause neurovascular effects in the tissues near to and following puberty.

Autonomic neural readjustments with emotional instabilities are prominent near puberty, menstruation, pregnancy and the menopause, periods in which otosclerosis deafness is prone to increase. They are also associated with endocrine variations, some of which, as I have pointed out, are known to be capable of affecting bone metabolism.[3] These factors intensify autonomic sensitiveness and are repeated or prolonged at these periods of life and during any severe emotional experiences at any time. What I am trying to say is that all this fits in perfectly with the known facts concerning age incidence and exacerbations of otosclerosis clinically and histologically observed.

Emotional sensitivity or instability may be due to the deafness, increased by it, or due to causes other than the deafness, whether present before or after deafness supervenes.

To learn whether a patient's nervous temperament differs from his or her siblings before the event of the deafness is often very difficult.

In these investigations it is pertinent to keep in mind that the teen-aged or adolescents who develop deafness from otosclerosis are in several respects in a different category from the child who is deafened from other causes:

1. He is seldom if ever deafened before his teens, and therefore
2. Not until after he has acquired a large vocabulary and all that this connotes.
3. His deafness does not actually come on suddenly, nor usually is it noted markedly in one ear alone.
4. There is seldom pain or any operative procedure associated with it, so that
5. He is deprived of the kind of concern and sympathy and understanding on the part of his parents and family that painful and operative illnesses engender.
6. Everyone is unaware of his deafness at first because it is slight and there is no apparent reason for any deafness.

7. On the other hand, unlike the deafmute or the severely deafened child, he is not subjected to overconcern and protection. He is not relieved of responsibility or from doing things for himself. On the contrary, he, and especially she, is subjected to increasing obligations and family duties.

8. As he grows older he is subjected to increasing irritations because he does not hear perfectly and therefore may appear stupid and inattentive. He becomes progressively frustrated, apathetic and retiring, with loss of attention in school and at home. He either assumes a "dead pan" expression, tends to withdraw from social contacts or manifests an exaggerated eagerness, or puts on an act or displays of temper. He may talk too much and monopolize the conversation so as to avoid having to listen to others. A common trait is to break into and interrupt conversations, without regard to the context of the subject under discussion; to keep talking largely to prevent trying to hear what others are saying, etc. Because he hears his own voice well, he may speak softly with the result that everyone tends to speak to him softly. All of these reactions really upset not only him *but his family and friends and accentuate his and their* emotional reactions.

I find that almost all persons having otosclerosis deafness reveal many characteristics showing that as a group they are emotionally hypersensitive, such as

1. Persistent search for a magical cure when they have been repeatedly informed by competent authorities that there is no cure.

2. Lack of adequate cooperation with their physician.

3. Failure to admit the need of lipreading or hearing aids, and often violent objections to same; a false sense of shame.

4. Willingness to submit to the fenestration operation even when told by several competent otologists that they are not favorable subjects for this procedure.

5. Exaggeration of slight improvement in hearing (even 5 db at one or two frequencies) following the fenestration operation.
6. Exaggerated depression following failure of operation.
7. Exaggeration of a tinnitus which you can show them is not really as loud as they would have you believe.
8. Flushing in one or both cheeks on slight provocation.
9. Sweaty or cold hands or feet.
10. Bringing with them a long list of written questions.
11. Failing to listen to what others are saying, even to the otologist whom they consult.
12. Paying more attention to what they themselves are saying or desire to say than to what their physician is saying or trying to find out.
13. Many of the other symptoms known to the neuropsychiatrist, or revealed by the newer mechanical and electronic techniques.

For many years I have been under the impression that in families with otosclerotic deafness the oldest or the elder of the siblings in each family were the ones most likely to be affected; also that in large families, and especially if there was a lapse of several years before the youngest was born, the youngest was frequently the one with otosclerosis. Being the oldest or the youngest usually entails a markedly different environment in the family and outside of the family, even in the uterus, than that encountered by the other siblings. In the case of females the oldest girl often takes on much of the duties of the mother, and all the social limitations which this entails, and the youngest girl, or boy, may be unduly pampered, overprotected and spoiled. Although responsibilities would generally be less for the younger child, this does not necessarily make for ideal emotional balance, and many times the youngest child is subjected to more disturbing emotional upsets than the older children. On the other hand, being the oldest furnishes a longer time in which the child can be subjected to emotional strains, and more time for an otosclerosis to develop.

With these ideas in mind I tabulated my observations in 224 cases of otosclerosis, with the following results: (In all instances where the exact ages or sequences were not obtained, this is indicated by a question mark (?) or the case is excluded from the calculations respectively.)

In females when there were two siblings, 72 per cent were the elder and 28 per cent were the younger.

In males when there were two siblings, approximately 50 per cent were the elder and 50 per cent were the younger (too few cases).

FEMALES		MALES	
Age of onset before 20	7	Age of onset before 20	6
Age of onset after 20	13	Age of onset after 20	8
Age of onset ?	1		
	21		14

In females when there were three siblings, 60 per cent were the eldest and 40 per cent were the youngest.

In males when there were three siblings, approximately 75 per cent were the eldest and 25 per cent were the youngest (too few).

Including those in the middle the distribution was:

FEMALES		IN FEW MALES EXAMINED	
Oldest	39.3 per cent	Oldest	66.6 per cent
Middle	33.0 per cent	Middle	16.7 per cent
Youngest	27.7 per cent	Youngest	16.7 per cent
	100.0 per cent		100.0 per cent

FEMALES		MALES	
Age of onset before 20	14	Age of onset before 20	3
Age of onset after 20	9	Age of onset after 20	6
Age of onset (?)	1	Age of onset (?)	2
	24		11

In the two and three sibling groups the ratio of females to males examined was roughly 2:1. In the low brackets of sibling number my impressions appear to have been substantiated.

When there were four siblings the distributions were about equal for all age sequences in males and females, and the age of onset before 20 and after 20 was about the same percentage.

When there were five or more siblings the distributions were:

FEMALES	
Oldest	13 per cent
Next to oldest	6 per cent
Middle	26 per cent
Next to youngest	18 per cent
Youngest	37 per cent
	100 per cent

IN THE MALES ABOUT THE SAME BUT TOO FEW

FEMALES		Per Cent
Age of onset before 20	11	17.7
Age of onset after 20	47	75.9
Age of onset ?	4	6.4
	62	100.0

MALES		Per Cent
Age of onset before 20	6	35
Age of onset after 20	10	60
Age of onset ?	1	5
	17	100

Only 13 were over 40 years of age when I first examined them. The ratio of females to males examined in the four and five sibling groups was above 3:1.

Herein there was a large percentage of younger (37 per cent) and next to younger (18 per cent) total 55 per cent; and middle placed siblings with otosclerosis (26 per cent), total 81 per cent; and a small percentage of next to oldest and oldest (19 per cent).

That the incidence was higher in the oldest than in the youngest, or younger in the smaller families was expected, but that the reverse woud be found in the larger families was unexpected.

In the higher sibling brackets my impressions were wrong except as to otosclerosis being frequently encountered in the youngest siblings; but it was not only frequent, it was almost three times that in the oldest of these siblings. Had I examined more patients in smaller families than in large families this unbalanced impression would be explainable, but the fact is that the reverse was true. I examined more patients in the larger families than in the smaller families. This at first thought seemed to mean that all of my work was wasted; however, I noticed that the eldest and the youngest taken together did constitute by far the greater number of siblings with otosclerosis in all the age sequence categories. I intend to examine a larger number of patients in the expectation that something more informative will be revealed, that the data will be more significant.

In order to use controls who also were subjected to the reactions caused by noninflammatory deafness I chose patients with nerve deafness. In these neural deafness patients in no category of sibling number was there a higher incidence in the younger than in the older siblings. This was to be expected because of the greater number of years lived by the older siblings; but it did not obtain in the large otosclerosis sibling groups, although it would be logical to expect the larger the number of siblings the older the eldest would be, and the longer time he would be exposed to the etiological factors.

One more observation: For the past three years I have examined the circulation in the arterioles and venules in the occular conjunctiva of all my patients and thus far without exception have noted a sludging of the blood, and certain other vascular phenomena in every patient with deafness from otosclerosis.

In two sets of identical twins, the one in each pair having typical otosclerotic deafness repeatedly revealed sludging of the blood phenomena, whereas the twin who had no deafness showed either no sludging of the blood, or distinctly less than the one with otosclerosis.

All these otosclerosis patients, although usually believing themselves in perfect health, were emotionally disturbed, hypersensitive people. This is one explanation for the sludging of the blood, because autonomic reactions often cause stasis in the organs and tissues of the body with sludging of the blood and related phenomena. The sedimentation rate, although it tends to correlate with sludging, was seldom increased in these patients. There is much more to this but I shall not go into it at this time. It is relevant to recall that all the theories now seemingly with the most plausible deductions from the observed pathology (and all such have been derived only from pathology) have been based on local congestion, stasis, endocrine and metabolic disturbances, or anoxia in the bony capsule or the neural tissues of the ear. All such theories have been derived only from morphological studies of the lesion, the dead end of the search. They do not explain the *cause* of otosclerosis. Not only is all of the terminal pathology of otosclerosis explainable by the hypothesis I herein set forth, but also a fundamental factor in etiology.

I submit that the immediate underlying cause of otosclerosis and any auditory neural deafness incidental to it is causable by autonomic dysfunction, stasis, anoxia, etc., as outlined in this paper. Whether it is one or the other or both of these disorders depends upon the capillary beds affected. No other explanation seems to me probable.

If my thesis is tenable, what can we do about it? Although in none of the older theories was there any hope for prevention or for control, now at least there is a glimmer of hope because emotional and behavior traits and reactions are in man susceptible to modification. Even a shortening of emotional reactions will help to some extent; moreover, liability and assets in the emotional nature may be assessed even as early in life as the preschool years, and appropriate medicinal and neuropsychological measures taken to improve defects. Such measures are indicated not only to insure a happier social life, but also a life without many of the ills otherwise impending. When there is an inherited or potential tendency to it, one of these ills is otosclerosis. Will anyone avail himself of such preventive measures? I wonder!

The approach I have outlined is beginning to pay dividends in such psychosomatic diseases as duodenal ulcer, diabetes and asthma. Why should not the otologist try to control the effects of emotional factors in otosclerosis?

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PLASMOCYTOMA: A CASE WITH ORIGINAL LESION ON THE EPIGLOTTIS AND METASTASIS TO THE TIBIA.*†

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The observation of a second case of plasmocytoma, whose primary tumor occurred in the upper respiratory tract, warrants reporting, with follow-up information of the first case.[1] The tumor in the first case was attached to the right base of the tongue by a broad pedicle, and in the second, to the rim of the epiglottis, extending to the adjacent pharyngeal wall. Both are associated with myelomas in distant medullary bone; however, neither have shown Bence-Jones protein in the urine.

The first case, age 63, diagnosed plasmocytoma, April 27, 1946, is symptom-free, working as a filling station operator. No radiation therapy was used on his bone lesions, which were ovoid areas of rarefaction in the right and left ilium. Films of cranium, chest, long bones and pelvis, made Jan. 10, 1951, showed none of these areas of rarefaction.

These two examples of plasma-cell tumor showing soft tissue and osseous lesions simultaneously have one early metastasis of the plasmocytoma to adjacent lymph nodes, and in both the appearance of the lesions in the medullary bone. Tumor activity within the hematopoetic system occurs in both cases, but no secondary anemia has been found. In Case 1 an adjacent cervical lymph node contained tumor, and in Case 2 a similar node was free of tumor. These are good examples of the paradoxical nature of every set of facts regarding plasmocytoma and multiple myeloma. Stout and Kenney[2] question the use of the word metastasis of this tumor, stating:

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"Indeed it is quite possible that all of these so-called metastases represent examples of multiple primary involvement, in which the nasal or oral involvement preceded the involvement of other sites, often by a long period of time. If the secondary involvement of bone marrow is an example of true blood-borne metastasis, it seems almost incredible that there should not have been at least some suggestion of lung involvement." Their report is a most complete review of 104 published cases of plasma-cell tumors of the upper air passages and oral cavity, adding seven new cases.

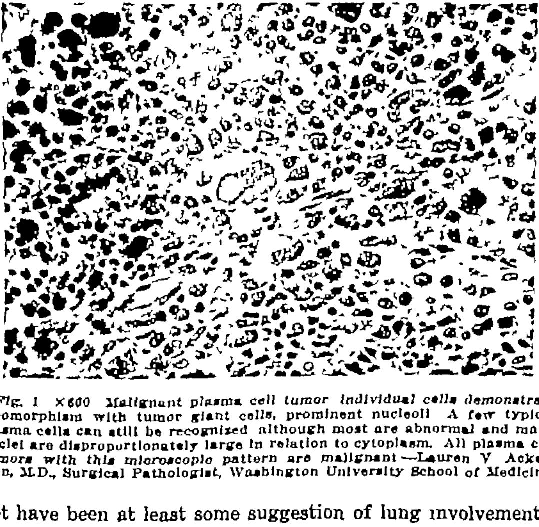

Fig. 1 ×*600* Malignant plasma cell *tumor Individual cells demonstrate* pleomorphism with tumor giant cells, prominent nucleoli A few typical plasma cells can still be recognized although most are abnormal and many nuclei are disproportionately large in relation to cytoplasm. All plasma cell *tumors with this microscopic* pattern are malignant —Lauren V Ackerman, M.D., Surgical Pathologist, Washington University School of Medicine.

SUMMARY.

A case of plasmocytoma is herewith added to the record whose primary or principal growth was located on the epiglottis and pharyngeal wall and associated with a large focus of myeloma within the tibia. Increase of plasma cells in the bone marrow and typical plasma cells in the tumor made the diagnosis, but the tumor shows atypical giant cells.

The responsibility of proper management rests on the laryngologist who sees the tumor first. It should be promptly removed and the area subjected to Roentgen therapy. The skeletal system is completely X-rayed to establish amount of dissemination.

The urine is checked for Bence-Jones protein, which, if present, indicates activity of tumor in the bone marrow.

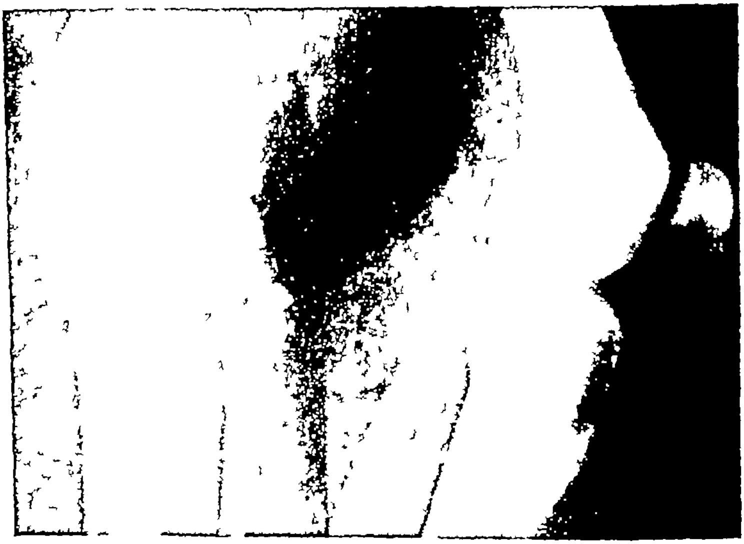

Fig 2 Anteroposterior and lateral views of the left knee demonstrate a *large metastasis in the lateral condyle of the tibia* The metastasis is characterized by a large translucent area involving most of the lateral condyle of the tibia with extension into the metaphysis This area is approximately 6 cm by 7 cm by 5 cm The margins of the defect are well defined, but they are slightly irregular and rough due to dissolution of the bone and its replacement by the tumor Metastatic tumors occurring in bones distal to the knee and distal to the elbow are quite rare This patient has not complained of pain in any other bones and a chest film made at this same date was negative for pulmonary metastases—Wendell G Scott, M.D, Associate Professor of Radiology, Washington University School of Medicine

Serum proteins are checked, since high globulins are associated with plasmocytoma. Anemia is common as the patient enters the terminal phase. Repeated sternal punctures are extremely important, increased plasma cells in the bone marrow showing presence of myeloma, thus altering the prognosis.

Broad field radiation is more desirable than interstitial elements. The case presented herewith is a good example of an isolated bone metastasis, apparently controlled by radiation, followed with curettement and repair of the bone defect with bone chips.

Case 2 G P R., white male age 52 was admitted to McMillan Hospital on Sept. 1, 1949, for removal of a round mass attached to the left border of the epiglottis. It was 2 cm in diameter and had the color and texture of lymphoid tissue A small palpable soft gland was noted at the angle of the left jaw

Surgery. On Sept. 2, 1949, the tumor was exposed under local anesthesia for the purpose of biopsy and removed with cautery This proceeded with ease, and just as the spatula was removed from the base of the tongue a copious hemorrhage ensued The exact area could not be found. Sponge pressure controlled it, but bleeding began again when taken away. At this point the left external carotid was tied, removing the enlarged lymph gland Laboratory findings, Sept. 1, 1949, were not significant: Urine negative, no Bence-Jones protein; R. B C 4 79, W B C 9,300, hemoglobin 98 per cent—15 2 Platelets normal, Schilling, bas. 2, eos. 2, stab. 1, seg 74, lym. 15, mono 6 The Kahn was negative.

Elaborate study was made of sections of the tumor, reported on Sept. 13, 1949 by Dr Ackerman and Dr Stephens as follows The surface exhibits stratified squamous epithelium which has been interrupted at one point, a fragment of cartilage opposite At this point there is necrosis of the underlying tissue The bulk of the tissue is composed of an extremely cellular mass which forms a rounded 'nodule' This is composed of cells which appear generally in somewhat irregular sheets or strands and which for the most part have a moderately abundant cytoplasm and eccentrically placed hyperchromatic nuclei in which there is often some typical chromatin element. Throughout the section there are numerous giant cells; some of these having a pattern similar to those cells described above, while others appear multinucleated and less distinctive."

He was discharged from hospital Sept. 8, 1949, before the complete pathological studies were returned Films of long bones, pelvis and chest were made on Sept. 12, 1949, by Dr. Wendell Scott, at which time radiation therapy was begun over the left cervical and jaw region and continued until Nov 12, 1949, 38 exposures totaling 6,450 r

Tenderness appeared over upper third of left tibia late in October, 1949; X ray on Nov 9, 1949, confirmed it as a metastasis (see Fig 2) He entered Jefferson Barracks Hospital, Nov 17, 1949, where a biopsy was made and this showed myeloma He returned to Dr Wendell Scott and between Dec. 12, 1949, and Jan. 7, 1950, received daily radiations over the upper third of the left tibia, totaling 3,750 r The area remained painful and daily treatment was again given via another portal, from Jan. 9, 1950, to Jan. 14, 1950, for a total of 550 r

Films of the area on Feb 24, 1950, and March 24, 1950, showed the area of metastatic tumor unchanged, and he was admitted to Barnes Hospital for study. Films of skull, chest, lumbosacral spine, cervical spine and pelvis were negative for metastasis. Repeated differential counts of the blood and sternal punctures on April 8, 1950, and April 11, 1950, showed normal erythroid and myeloid elements, but an increase in plasma cells, unevenly distributed in the marrow, some being young forms. This increase of plasma cells, together with the positive diagnosis of plasmocytoma in the original tumor, made a likely diagnosis of multiple myeloma possible.

Surgery: On April 15, 1950, the lateral aspect of the tibia was exposed by Dr. David Oliver, and a large cavity which represented the tumor was curetted, repacked with rib bone from the bone bank. Multiple sections of the tissue showed no definite evidence of persistence of tumor, indicating a high radiosensitivity of the neoplasm. It was the certain opinion of Dr. Ackerman that the tumor is present in other locations, and that the prognosis is hopeless. Palliative effort is advised when symptoms again appear.

He was discharged from Barnes Hospital, April 23, 1950, improved, with the left knee stiff but not painful. He remained the same when seen Dec. 14, 1950.

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A GIANT RHINOLITH.*

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Although over 300 cases of rhinolith have been recorded,[1] the occurrence of enormous nasal concretions is comparatively rare. Melay,[2] Myerson[3] and Gorman[4] extracted nasal calculi that weighed 85 gm., 56 gm. and 70 gm., respectively. The largest calculus recorded in the literature weighed 110 gm. and required Rouge's sublabial operation for its removal.[5]

The aim of this paper is to describe a nasal calculus which weighed 116 gm. (6 gm. heavier than the largest recorded) and which had greatly distorted the facial bones, causing symptoms suggesting a malignant growth.

REPORT OF A CASE.

S. H., a 50-year-old Arab male, was admitted to the ear, nose and throat service on Nov. 20, 1950, complaining of bilateral nasal blockage, excessive tearing from the right eye and foul discharge from the right nostril for the past 10 years. During the last two years he had noticed a gradual displacement of his right eye, a fullness of his right cheek and widening of the bridge of his nose. He did not complain of pain or epistaxis.

Examination revealed a marked outward and downward displacement of the right eyeball and an evident swelling of the right side of the nose and adjacent cheek region. A stony hard mass could be felt springing from the medial wall of the right orbit. The right nasal cavity was occupied by a large stony hard mass. A thick grayish discharge came from around the mass and had a foul, pestilential odor. The left nostril was obstructed by a displacement of the septum to that side. Posterior rhinoscopy showed a grayish mass occupying the right side of the nasopharynx and extending to the left, leaving a small slit-like opening in place of the left choana. There was no sign of the posterior edge of the nasal septum. Both nasal bones and also the nasal process of the left maxilla were displaced outward and to the left. X-ray study by Dr. G. Saleeby revealed a large irregular body occupying the nasal cavity

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and encroaching on the right maxillary and ethmoidal sinuses and on the right orbit. It was either a rhinolith or a bony mass; more probably the former.

Attempts to break up the mass or take a biopsy were unsuccessful on account of its hard consistency. An external operation to deliver it was, therefore, found necessary.

The operation was performed under pentothal, nitrous oxide and oxygen intratracheal anesthesia An incision was made from the inner extremity of the right eyebrow descending along the nasobuccal groove to the side of the ala nasae. The bone was exposed and divided according to Moure's lateral rhinotomy technique, thus obtaining a large opening through which the mass was grasped and, after considerable difficulty,

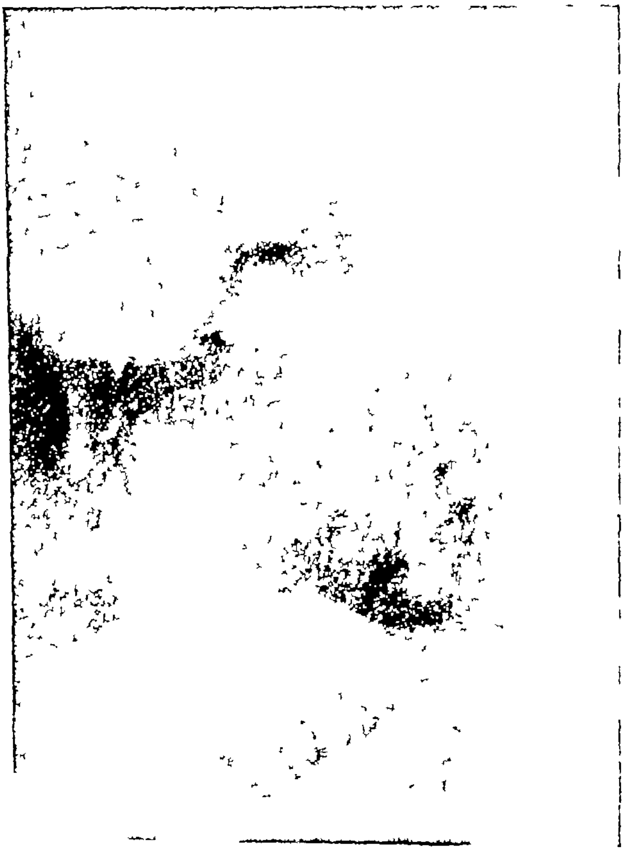

Fig 1 Roentgen film of lateral view of nose showing rhinolith in situ

dislodged and extracted. The mass was ivory-hard, grayish black, and presented a comparatively smooth, rounded lateral surface which fitted snugly into the right maxillary and ethmoidal sinuses, and a medial (septal) rough surface which had fractured and pushed the septum against the lateral wall of the left nostril. It measured 7 cm. by 6.2 cm. in its greatest diameters and weighed 116 gm. (see Figs. 1 and 2).

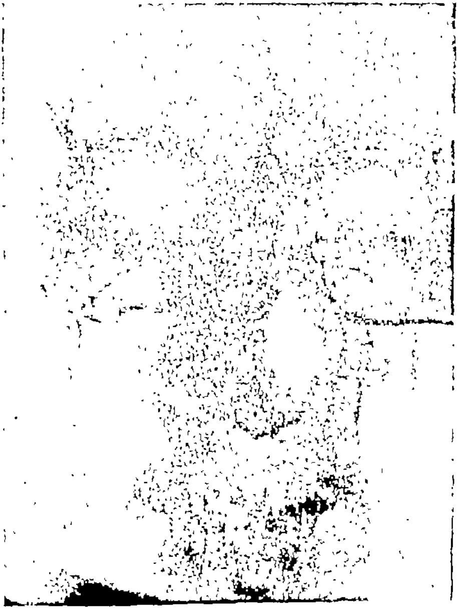

Fig. 2. Roentgen film of anteroposterior view of skull before operation. Note encroachment of rhinolith on right maxillary and ethmoidal sinuses and its extension into the right orbital cavity.

Inspection of the nasal cavity now revealed complete absence of the lateral wall of the right nostril (the turbinates, the nasal surface of the maxilla, the ethmoidal labyrinth, the orbital plate of the ethmoid bone and the lacrimal bone). Only part of the anterior cartilaginous septum had remained and this was replaced centrally by a septum forceps. Like-

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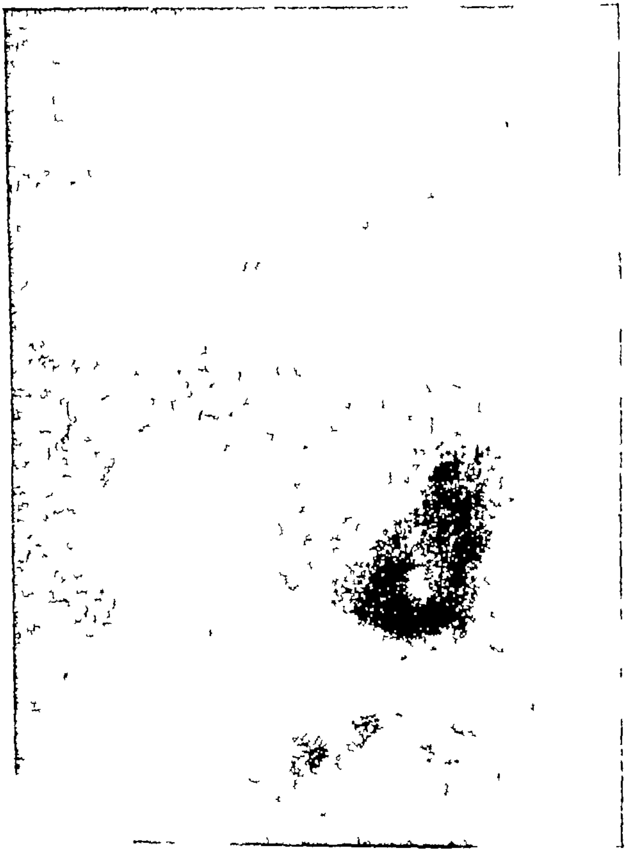

Fig 1 Roentgen film of lateral view of nose showing rhinolith in situ.

wise, both nasal bones were fractured and pushed back in place The nose was cleansed as much as possible and packed with iodoform gauze to control bleeding The external wound was closed with cotton sutures

Two days later the packing was removed Penicillin therapy was started on the day of operation and continued for nine days. The postoperative course was uneventful and on Dec. 6, 1950, 12 days after operation, the patient was discharged as recovered

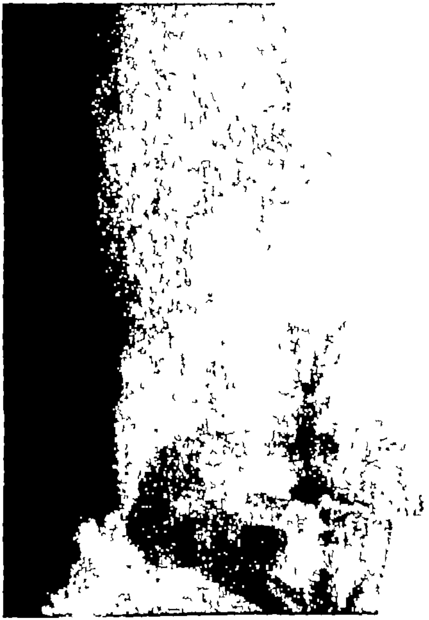

Fig 5 Roentgen film taken 10 days after removal of the rhinolith. Lateral view

Chemical analysis (No S-381-50) of the rhinolith by Prof. W. A West revealed the following Organic matter plus moisture, 33 per cent, calcium phosphate, 43 7 per cent, calcium carbonate, 21 8 per cent, undetermined, 1 5 per cent.

SUMMARY.

1. Large rhinoliths are rare.

2. A rhinolith weighing 116 gm. (6 gm. heavier than the largest recorded) and simulating a malignant growth of the nose is reported.

3. A radical external operation was required to remove it.

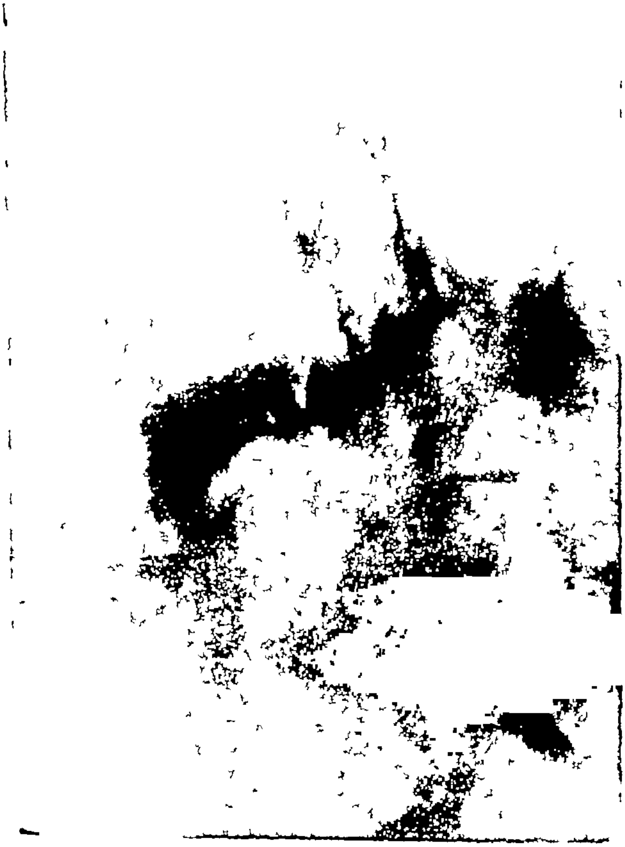

Fig 6 After removal of rhinolith, anteroposterior view

4. Photographs of the rhinolith and also preoperative and postoperative Roentgen films of the patient are presented.

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The first annual meeting of the Eye, Ear, Nose and Throat Hospital Alumni Association will be held in New Orleans, La., April 26, 27 and 28, 1951. All physicians who have received their specialty training at the hospital are cordially invited to attend.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March 1, 1951.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

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Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.

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Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.

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Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.

Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

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Aurex (Semi-Portable).

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Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

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NEURAL MECHANISMS OF HEARING: SOME EXPERIMENTAL STUDIES OF THE AUDITORY NERVOUS SYSTEM.[1]*

W. D. NEFF, Ph.D. (by invitation),
Chicago, Ill.

I. Introduction.

Theories of the neurophysiological basis of hearing have been based primarily upon results of anatomical and physiological investigations. A complex system of neural tracts and nuclei has been described in some detail, and observations have been made on the flow and interaction of nerve impulses in this system. Although both kinds of evidence, anatomical and neurophysiological, are still incomplete, they have been sufficient for the formulation of fairly elaborate hypotheses as to how such a system might mediate "hearing"—the name we usually use to refer to behavior which is initiated or guided by sound stimuli.

As a further step in the development of a theory of hearing, we need another kind of evidence, the kind that comes from experiments which are sometimes labeled psychophysiological, or more simply, "behavioral." These are experiments

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[1] The experiments cited in this paper were supported in part by the Office of Naval Research, Contract N6 ori-20, T.O. XXIV, ONR Project NR 140 608.

*Read by invitation at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., at Cleveland, Ohio, Jan. 15, 1951.

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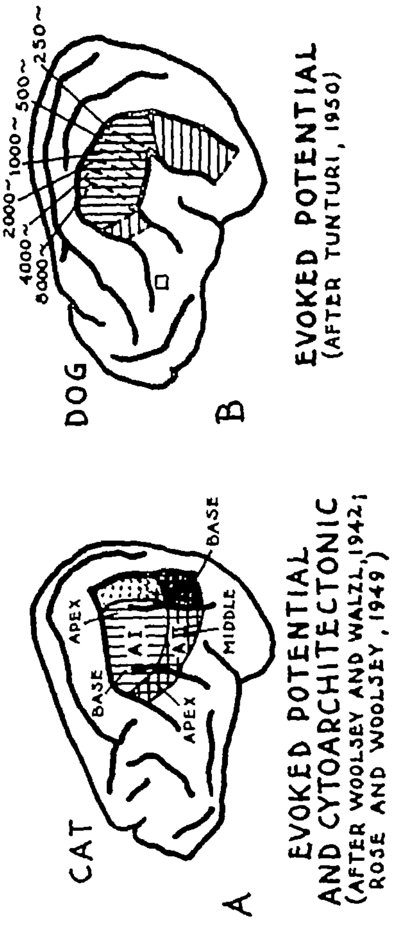

Fig 1 Maps of the auditory cortex

in which the nervous system is manipulated (*e.g.*, by section of a tract or ablation of a nucleus) and the effect upon some auditory discrimination is measured.

In the present paper, two examples will be given to illustrate *1.* how psychophysiological experiments are suggested by the hypotheses formulated from anatomical and neurophysiological evidence, and *2.* how the results of the experiments may lead to revision of theory concerning the neural mechanism of hearing.

II. The Discrimination of Changes in Frequency.

From a number of experiments there is rather clear evidence that the cochlea is projected upon the cerebral cortex in a systematic fashion (Woolsey and Walzl, 1942; Licklider, 1941; Tunturi, 1944, 1950).

In Fig. 1 maps of the auditory areas of the cortex of the cat and dog are shown. Woolsey and Walzl mapped the auditory areas of the cat in the following manner: In an anesthetized animal, the cochlea was opened so as to expose the nerve endings along the spiral lamina. At different points from base to apex, these endings were stimulated by weak electric shock. At the same time the potentials evoked at the cortex were recorded. In this way two auditory areas (see AI and AII, Fig. 1) were plotted. In AI, the base of the cochlea was projected anteriorly in the ectosylvian gyrus and the apex, posteriorly. In AII, which extended from the anterior ectosylvian across the pseudosylvian and into the posterior ectosylvian gyrus, the apex of the cochlea was projected anteriorly and the base, posteriorly. In the original experiment, area AI was shown as extending to the postsylvian sulcus. Later, Rose and Woolsey (1949), on the basis of both cytoarchitectonic and electrophysiological results, produced the map as shown in Fig. 1A, in which a third, posterior ectosylvian area is shown in addition to areas I and II.

In a series of studies on the dog, Tunturi obtained results which may be summarized briefly by the map of Fig. 1B. He, also, used an evoked potential technique but stimulated the determine the difference limen (DL), that is, the smallest discriminable frequency change. After bilateral ablation of auditory areas I and II of Woolsey and Walzl, the cat relearned the 200-2,000 c.p.s. discrimination, and, upon further training, was able to make a discrimination of 200-250 c.p.s. and finally 200-205 c.p.s.

Cat 2, trained preoperatively to a difference of 50 c.p.s., was able *to perform at the same level postoperatively.* The preoperative record of Cat 3 is shown for comparison with Cat 1. A difference limen of 5 c.p.s. was found for Cat 3 preoperatively. This is the same as the postoperative difference limen for Cat 1.

On the basis of these results, which must be considered tentative until further experiments are completed, the hypothesis that the auditory areas of the cat are essential for discrimination of frequency must be rejected.

III. Localization of Sound in Space.

In discrimination requiring the localization of sound in space, the interaction of neural impulses initiated at the two ears is of primary importance. Anatomical evidence suggests that this interaction may take place at any of several levels of the nervous system — cortex, inferior colliculus, superior olivary complex, primary cochlear nucleus, or perhaps even at the level of the spiral cochlear ganglion (see *e.g.,* Ades, 1944; Rasmussen, 1946). The effect of interaction has been measured by electrophysiological methods at the cortical level (Rosenzweig, 1950), at the inferior colliculus (Ades and Brookhart, 1950), and at the cochlea (Galambos, Rosenblith and Rosenzweig).

Neither the anatomical nor electrophysiological evidence answers the question: At what level can the afferent signal lead to the appropriate motor behavior, the localizing response to a sound stimulus? Again behavioral evidence is necessary.

A series of experiments has been done in which the ability of cats to localize sound has been measured before and after ablation of parts of the auditory nervous system (Neff and

Yela, 1948; Arnott and Neff, 1950; Neff, Arnott and Fisher, 1950). The animals were trained to approach and open the door of one of two boxes in order to obtain food reward. The correct choice on a given trial was indicated by a sound stimulus (buzzer) placed behind one of the boxes. The angle between successive positions of the buzzer as measured from a starting cage could be varied from 180° to 0°.

No change in ability to localize was found after unilateral ablation of the auditory areas I and II. An unmistakable change in performance appeared, however, after bilateral removal of these areas. Normal animals are able to make correct discriminations when the angle between successive positions of the sound stimulus is as small as five degrees. Animals without auditory cortex reach the same criterion of performance only for angles of 40 degrees or greater. The behavior of operated animals was also more variable than that of normals. A high percentage of correct responses was not maintained over a long period of time even when the angle to be discriminated was very large.

It is concluded that the auditory cortex is not essential for discriminations which involve the localization of sound in space; it is necessary, however, for the most effective utilization of sound cues in guiding such behavior.

The experiments cited above were done in collaboration with R. A. Butler, G. P. Arnott, J. D. Fisher and M. Yela.

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HEADACHE AND AUTONOMIC IMBALANCE.*

RAYMOND L. HILSINGER, M.D.,
Cincinnati, Ohio.

Headache is one of the commonest complaints encountered in medical practice. It is usually the symptom in a syndrome; therefore, a physical examination and a complete history are necessary for differential diagnosis. Vascular headaches, most usually typed as migraine, because of a definite mechanistic pattern, can be classified as a definite clinical entity. Lennox[1] has conservatively estimated that there are six million migraine or migraine type sufferers in the United States, which probably includes the entire classification of vascular headaches. Others estimate as many as ten million. Vascular headaches include classical migraine, tension headaches, migraine equivalents, histamine cephalgias, and a group of unclassified vascular headaches, all of which challenge the diagnostic ability of the practicing physician.

The acute attack of vascular headache is now adequately controlled with such vasoconstrictors as ergotamine tartrate, dihydroergotamine, and oral ergotamine tartrate with caffeine alkaloid. Horton,[2] Alvarez,[3] Ryan,[4] Hansel,[5] Friedman,[6] Wolff[7] and Hilsinger,[8] among others, have reported excellent results ranging from 80 to 90 per cent with all or some of these drugs. It must be pointed out that up to the present these drugs are only effective in the acute attack. Employing them, especially Cafergot, for interval therapy will not reduce the incidence or frequency of attacks.

It is the purpose of this paper to report the use of a drug which, in our hands, has been found effective in prolonging

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*From the Department of Otolaryngology, College of Medicine, University of Cincinnati School of Medicine.

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the quiescent period between acute attacks of vascular headaches. The need of such therapy is indicated because:

1. vascular attacks do not always respond promptly to vasoconstrictor medication.
2. the taking of an ergot preparation over long periods is not always desirable,
3. some of the medications employed result in undesirable side effects as nausea, cramps, vomiting, etc. Such effects present psychologic hazards to the patients so that they become somewhat resistant to medication, and subsequent attacks are more difficult to control.

Friedman[8] has stressed the personality and emotional conflict aspect of these headaches. The most common precipitating factors, *i.e.*, worry, fear, anger, etc., place strain on the autonomic nervous system. For this reason, interval treatment must be aimed at removing these, usually by psychotherapeutic means. Sufficient rest and removal of undesirable environment stimuli has been recommended and found effective.

Drug therapy is also indicated if it can be directed towards dampening the responses of the autonomic nervous system. The use of such a drug enhances greatly the psychotherapeutic measures and enables the patient to adjust his living in a quieter and easier fashion. We have found that the following combination fulfills this purpose:*

0.3 mg. ergotamine tartrate—sympathetic inhibitor
0.1 mg. Bellafoline—parasympathetic inhibitor
20 mg. (1/3 gr.) phenobarbital—central sedation

Rothlin,[9] Jores and Goyert,[10] and Tod and Jones[11] reported on the rationale for and the results with this combination on quieting the autonomic nervous system. This particular combination apparently acts in synergism and is effective in reducing the intensity of nerve response to stimulating experiences. Moore,[12] Slagel,[13] Karnosh and Zucker,[14] Harris[15] and Nelson[16] all reported beneficial clinical results from its use.

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*Manufactured under the trade name of Bellergal, Sandoz.

RESULTS.

Twenty-five patients are reported in this series. Admittedly, a larger series would be desirable but, because of the criteria used in selecting these patients, this was difficult to attain. Only those patients with frequently recurring attacks who did not respond to the more common vasoconstrictor drug therapy, or those who complained of pronounced side effects from these drugs, were selected.

Dosage of this drug must be adjusted to the individual needs of each patient. The usual recommended dosage is four tablets per day — one 15 minutes before each meal and one at bedtime. This dose can be increased to five or six tablets when these individuals are under greater stress. The maximum dose is given at the beginning of the therapeutic regimen and then gradually is decreased as improvements appear. Finally, some individuals will require only one tablet daily as a maintenance dose. Occasionally the larger dose may produce some drowsiness, in which case dosage must be reduced. Although Slagle[13] reports no deleterious side effects from this combination after prolonged use in 217 patients, prolonged use of any sedative is usually not recommended. Patients may be advised, therefore, to interrupt treatment two days out of every week, especially when maintained on a high (four to six tablets) dosage level. A similar recommendation is made by Harris[15] who suggests interrupting therapy with this combination one week out of four. This recommendation is made to avoid any possibility of habituation caused by the phenobarbital component.

Clinical results and outstanding case histories follow. Results for the entire series can best be seen in Table 1. A few case histories were summarized in order to show the variety in dosage. This points out again the necessity of individualized adjustment in order to obtain maximum effect from this type of therapy.

Case 2 G M, female age 23 Typical migraine since puberty with complete cessation during pregnancy The migraine attacks occurred every two or three weeks Cafergot and DHE-45 gave no results One Bellergal was given 15 minutes before each meal and another was given at bedtime several days before the impending attack. The patient is now completing her twenty fourth week with one mild migraine attack, and for the last few weeks has been maintained on one tablet per day

Case 6 M B, female, age 41 This case was diagnosed as histaminic cephalgia The headaches occurred every three to four days. For the past year the frequency of attacks has been notably reduced as a result of histamine desensitization however the patient complained of a tension type headache which occurred every three to four days to one per month Bellergal therapy was initiated and the patient is now completing her fifteenth week completely free of all headaches.

Case 7 H G., male age 29 Gave a history of grand mal epilepsy which has been completely controlled for the past two years with 3 gr of phenobarbital daily, however he continued to suffer with a tension type headache which formerly followed his epileptic seizures. These incapacitating headaches occurred weekly and would last for from four to 12 hours. Cafergot gave poor results Nicotinic acid gave moderate relief. Bellergal therapy was initiated along with nicotinic acid and the patient has been completely free of headaches for the past 14 weeks

Case 10 M M female age 68 Typical migraine since puberty Headaches increased in severity since menopause 16 years ago The headaches occurred regularly at two-week intervals lasting for 48 hours, during which time the patient had to go to bed Cafergot, Gynergen and DHE-45 gave no relief Nicotinic acid gave mild relief Bellergal therapy was initiated One tablet was taken 15 minutes before each meal Another tablet was taken at bedtime several days before the impending headache. The patient is now concluding her eighteenth week with one attack of migraine

Case 11 P G male age 37 Complained of a dull throbbing unilateral incapacitating headache. The patient had no aura and complained of nausea during the attack. Headaches were irregular in frequency occurring once in three to four days to one every two weeks. The headaches lasted from 24 to 30 hours. The patient gave a family history of headaches. Case was diagnosed as a migraine equivalent. Cafergot, DHE-45, codeine and nicotinic acid were of little or no value. Interval treatment with phenobarbital was also of no help The patient actually lived in fear of these headaches. Bellergal therapy of 1 q i d. was initiated and the patient is completing his seventeenth week with one very mild headache

Case 14 L. G male age 33 Frequently recurring headaches since 30 years of age Headaches were of comparatively short duration and occurred during early morning The case was diagnosed as a typical histaminic cephalgia. Nicotinic acid gave moderate results, and good results were obtained with Cafergot, however because of the frequency of the attack, it was felt that this patient was receiving too large an amount of ergotamine tartrate The patient was placed on one Bellergal tablet 15 minutes before each meal and he is completing his ninth week completely free of headaches. Of special interest is the fact that after about the fourth week the patient voluntarily offered the information that the symptoms from his duodenal ulcer have also been greatly relieved and that he had stopped going to his stomach doctor"

Case 17: L. B., female, age 55. Complained of headaches since puberty. Intelligent executive secretary. This case was diagnosed as a typical migraine. The headaches were so severe that she had to stay home three to four days each month. The headaches occurred every one to two weeks. Bellergal therapy was initiated and, to date, the patient has had two mild attacks of no consequence in 26 weeks.

Case 18: R. G., male, age 48. Tension headache and nasal allergy. Cafergot, DHE-45 and interval phenobarbital and antihistaminics gave poor results. Headaches occurred every three to four days, to one every two weeks. Bellergal, 1 t.i.d., was given, and 1 q.i.d. on days when the patient encountered added strain. At this time the patient has been completely free of headaches for 14 weeks. During the last several weeks the patient has adjusted his own dosage so that no medication is taken on certain days, with a maximum of four on others.

TABLE I. CLINICAL RESULTS.

	Name	Age	Sex	Diagnosis	Frequency of Attack	Length of Attack (Hours)	Duration of Bellergal Therapy (Weeks)	Number of Attacks since Bellergal Therapy
1.	L.F.	37	F	Tension	1/3-4 days 1/1-2 wks.	12-48	17	None
2.	G.H.	23	F	Migraine	1/2-3 wks.	48	24	1 Mild
3.	J.M.	32	M	Tension	1/2-3 wks.	8-12	12	None
4.	E.V.	45	M	Migraine Variant	1/2-3 wks.	4-8	14	2 Mild
5.	T.B.	40	F	Tension	1/2-3 days	8-24	14	1
6.	V.B.	49	F	Tension	1/4-5 days	8-12	15	None
7.	H.G.	29	M	Tension	1/wk.	4-12	14	None
8.	R.S.	46	M	Tension	1/2 wks.	8-12	29	4
9.	D.M.	35	M	Tension	1/2-3 wks.	12-24	8	1 Mild
10.	M.M.	68	F	Migraine	1/2 wks.	48	18	1
11.	P.G.	37	M	Migraine Equiv.	1/3-4 days 1/2 wks.	24-30	17	1 Mild
12.	M.R.	72	F	Hist. Ceph.	1/wk. variable	1-2	10	None
13.	C.R.	37	F	Tension	2/wk.	8-24	8	2/wk. Possible slight improvement.

TABLE I. CLINICAL RESULTS (Cont.).

	Name	Age	Sex	Diagnosis	Frequency of Attack	Length of Attack (hours)	Duration of Bellergal Therapy (Weeks)	Number of Attacks since Bellergal Therapy
14.	L.O.	33	M	Atypical Hist. Ceph.	1/3-4 days variable	1-2	9	1 Mild
15.	M.S.	36	F	Tension	1 wk.	24	22	None
16.	M.A.	28	F	Tension	1/1-2 wks.	8-10	15	None
17.	L.B.	55	F	Migraine	1/4 days 1/2 wks.	12	26	2 Mild
18.	R.O.	48	M	Tension	1/5 days 1/2 wks.	24	14	None
19.	E.H.	37	M	Tension	1/2-2 wks.	12-24	24	2
20.	L.M.	35	F	Tension	1/2-3 days	2-8	15	4
21.	W.T.	56	F	Tension	1/2 wks.	4-8	10	None
22.	L.O.	51	F	Tension	1/1-2 wks.	8-12	8	None
23.	L.G.	44	F	Tension-accentuated at menstruation	1/2 wks.	4-12	11	1/2 wks. No results.
24.	C.W.	50	F	Tension	1/wk.	8-12	17	4
25.	M.E.	26	F	Migraine variant	1/2 wks.	8-12	11	1

SUMMARY.

1. Autonomic imbalance is a major contributing factor in vascular headache.

2. During the acute attack, good results may be obtained with ergotamine tartrate, dihydroergotamine or "Cafergot."

3. Rest and removal of undesirable environmental stimuli is the preferred method of prolonging the quiescent period of frequently recurring vascular headache. Where this is impractical, a drug which will dampen the effects of the undesirable nerve impulses to the autonomic nervous system is recommended. A combination consisting of ergotamine tartrate (sympathetic inhibitor), bellafoline (parasympathetic inhibitor) and phenobarbital (central and subcortical sedative), (Bellergal), has been found to be effective for this purpose.

TABLE II. SUMMARY OF CLINICAL RESULTS.

TYPE HEADACHE	NO. CASES	EXCELLENT*	GOOD**	POOR***
Migraine	6	4	2	
Tension	17	11	1	5
Histaminic Cephalgia	2	2		
TOTAL	25	17	3	5
% Results		68%	12%	20%

*Complete relief, or a maximum of one attack.
**Noted reduction in number of attacks.
***Little or no relief.

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have stimulated others to use the method, as several papers have appeared since, with some modifications.

I, too, have modified it slightly, but basically it is Ruddy's operation; however, it has been my good fortune to operate on seven successful cases, and I feel that this warrants stumping for the procedure.

The approach offers advantages of attacking the problem under direct vision, and not by blind mutilating surgery. To illustrate this I might cite one case of a four-year-old boy with unilateral choanal atresia, first treated by the intranasal route with perforator and rasp. This opening soon closed and I did the second operation by a transpalatine approach. Under direct vision the posterior nasal fossa was one solid band of scar tissue extending forward to almost the middle of the nose; certainly over a much greater area than the original obstruction. With all this scarring even the transpalatine operation was not at first successful, and the palate was reopened and a skin graft was placed around a central sponge core. This second procedure insured the patency of the choana. I feel that observation of this condition well illustrates why so many done by the intranasal route are doomed to failure. Passing instruments through the narrow passage in the obstructed side of the nose creates raw surfaces for synechiae to form over in much greater area than the original obstruction.

The transpalatine approach gives greater insight into the problem at hand and in doing seven cases certain things became consistently evident: first, the nasal cavity on the obstructed side, especially posteriorly, is quite narrow (see Fig. 1-A). This is not due to a deviated septum but to a crowding of the lateral wall medially with a sweeping in of the lateral and posterior pharyngeal walls, thus shortening the anterior-posterior plane in the pharynx. Stinson's[14] article, in 1932, is the only one I have noted which mentions this finding, although Wright, Shambaugh and Green selected a transantral approach because of the narrowing of the nasal cavity on the obstructed side. Because of the above findings, the obstructing membrane is angulated to about a ten-four o'clock position when observing it through the palate (see Fig. 1-B). This is not shown in most drawings where the membrane is usually shown in the transverse position. It is this shortening of the lateral and posterior wall of the pharynx on the obstructed side, with the crowding of the lateral nasal wall, which makes removal of part of the vomer necessary for success of the operation. Because of the above deformity, even after operation, when probing the opening, the probe will follow downward and medially to drop into the pharynx instead of straight back as in the case of a normal choana. A higher palatal arch on the obstructed side is practically always present.

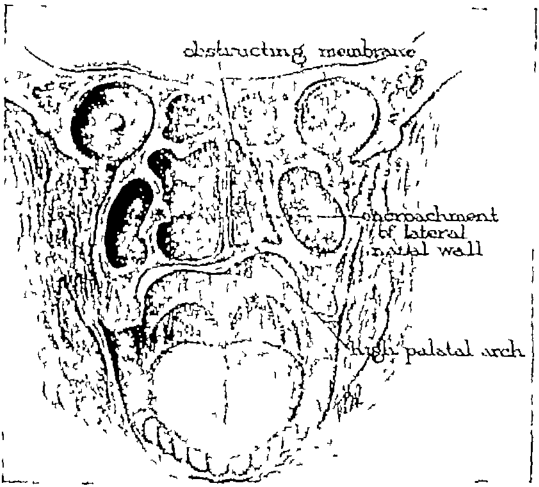

Fig 1-A. Coronal view at the level of the choanal obstruction on the left. Note the palatal elevation and the narrow nasal cavity

Why such deformities exist is not clear. The sphenoid body and medial plates of the pterygoid processes along with the septum and palatal bone form the choana. It has been shown by McGovern[11] that sinus and accessory auditory development

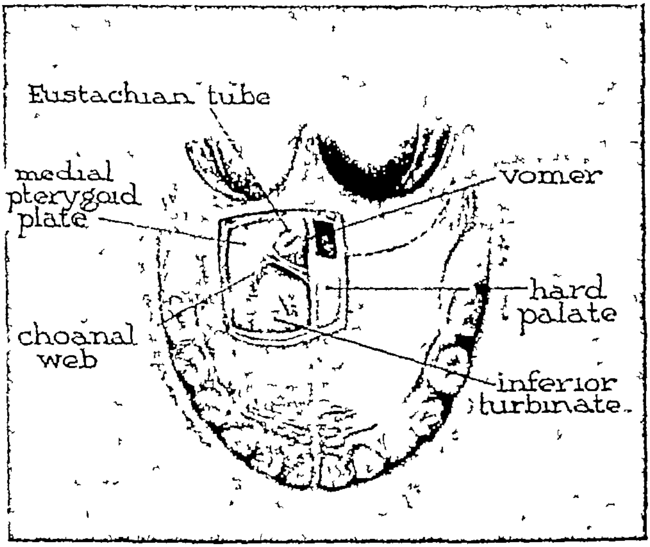

Fig 1-B Exploded view showing the crowding of the lateral nasal wall and the shortened nasopharyngeal cavity on the obstructed side

are normal. An X-ray of one of my cases (see Fig. 2) shows normal development of the sinuses even though the nasal fossa is quite narrow on the obstructed side. This might suggest that the theory of medial overgrowth of the vertical and horizontal palatal processes as an etiologic factor is a good one; however, as the maxilla, sinuses and Eustachian tube are known to be well developed, it would seem to me that lack of development could be a mechanical one due to the restriction of the obstructing web, whether bony or membranous. For this reason, it would seem a logical argument to operate on these cases early. From a technical standpoint this can be done easily at a very early age by the transpalatine approach. This brings up the argument that removal of so much tissue may in itself retard development. In answer to this, I must state that a year after surgery I have palpated the posterior border of the hard palate, and it is hard to discern any difference in the level of the operated and unoperated sides Removal of a good portion of the vomer doesn't seem to interfere with development of the nose as sometimes supposed, and oral surgeons not infrequently remove a section of the vomer to force back the premaxilla in cases of harelip. My first case, done at six months, and now five years postoperative, has normal nasal and facial development

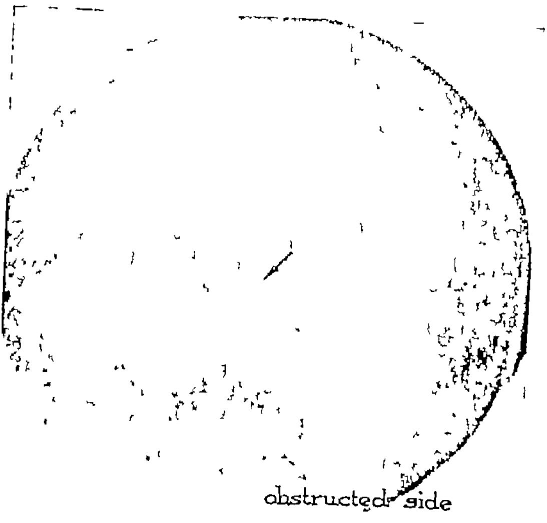

Fig 2. X ray showing choanal obstruction on the left side

Many otolaryngologists are loath to enter the palate, but I assure you it is an easy and safe procedure. For those of you who are not familiar with the technique or have forgotten, I will describe it and show you my modifications.

Ether anesthesia is given through an intranasal catheter on the obstructed side. An incision is made in a semicircular manner from just inside the second molar tooth well forward to just a centimeter in front of Scarpa's foramen (see Fig. 3). It will be seen that this incision extends much farther forward than Ruddy's original incision and also more laterally to include the greater palatine vessels in the flap. The incision is carried down to bone. This larger flap seems to allow for better healing, as all the cases have healed by primary union in a few days without slough. Inclusion of the arteries in the flap insures a good blood supply. This flap also does away with any fear of resulting perforation if one finds it

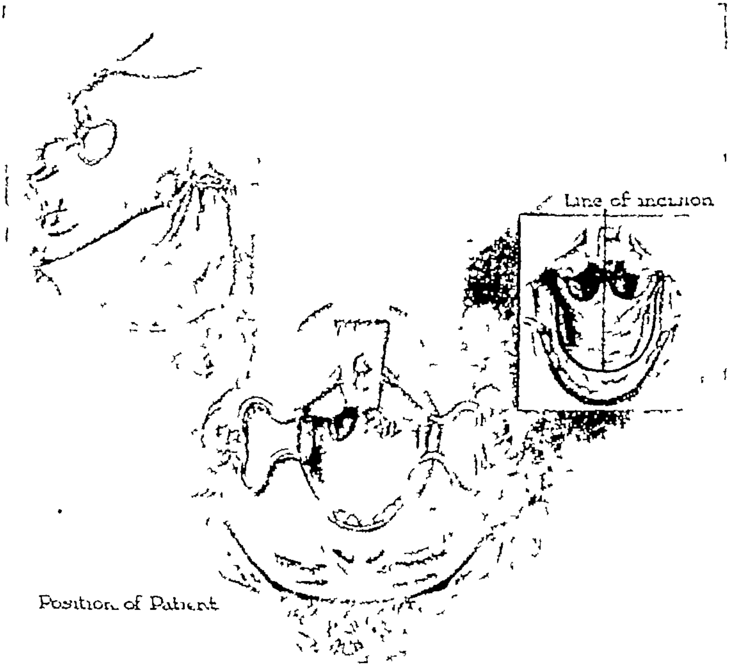

Fig 3 The procedure is done standing at the head of the patient and the incision starts from the second molar area and extends around to 1 cm posterior to Scarpa's foramen

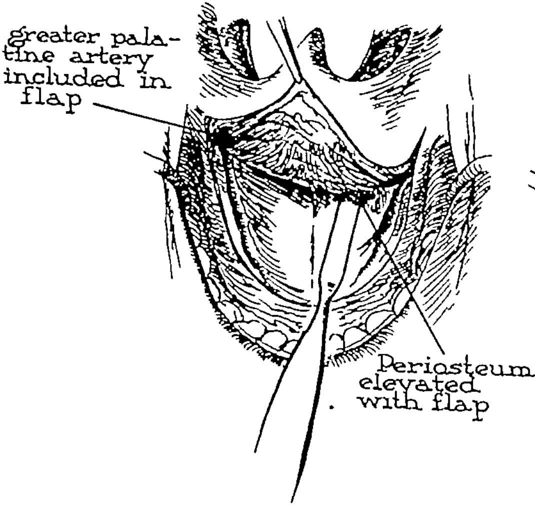
greater palatine artery included in flap
Periosteum elevated with flap

Fig 4-A Elevation of palatal flap Note vessels in relief on under surface of the flap.

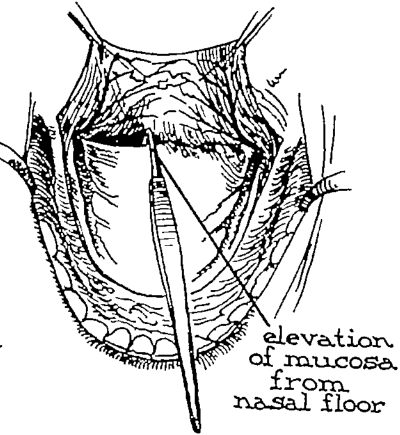

Fig 4-B Right angle Freer elevator freeing mucosa on the nasal floor

ahead of the obstruction with a gouge so that one gets a view on either side of the obstruction, as suggested by Ruddy.

After viewing obstructing plate one proceeds to bite it out clean to the margins with rongeurs (see Fig. 6). Goodhill[8] recently did a case by the transpalatine approach and he used a burr to take down some of the lateral wall in the region of the pterygoid bone. This appears to be a good adjunct in the technique to increase the opening by dealing with the crowding lateral wall. I intend to try this on my next case.

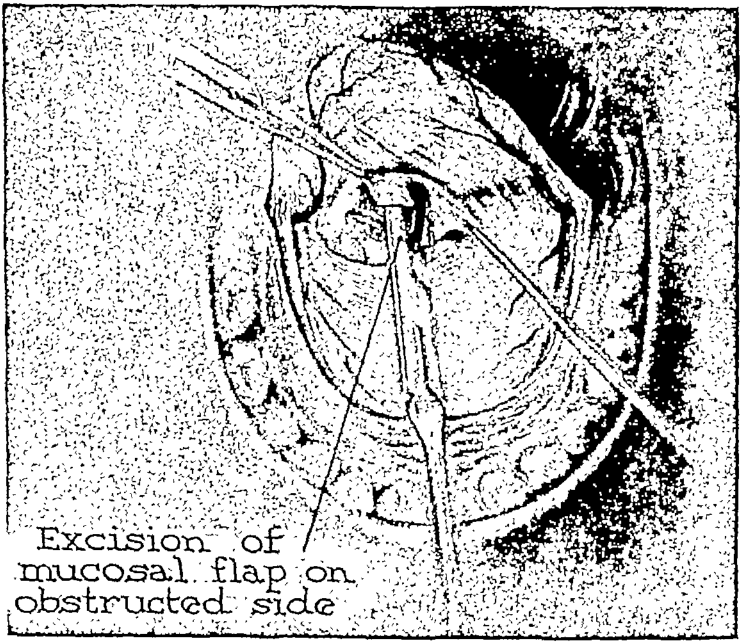

Fig. 7. Septal mucosa flaps separated and vomer bone removed. The septal mucosa on the obstructed side is being removed.

Now the septum has to be dealt with. I carry my removal of the palate slightly over the midline so as to expose the vomer attachment to the palatine crest. Here again, I vary the technique of Ruddy slightly. Before cutting the septum, I incise the mucosa along the posterior border of the vomer and then separate the mucosa on either side as in a submucous resection. The bony septum is then removed as much as is deemed necessary for an adequate opening. The mucosal flap on the operated side is then removed and the opposite flap is severed at the anterior and posterior margins of the septectomy (see Fig. 7). This creates a flap which can be laid over, covering the raw surface at the obstructoin site (see Fig. 8). If necessary the mucosa around the obstruction site can be denuded some to receive the raw surface of the flap. This flap is held in place with interrupted 00000 chromic catgut (see Fig. 9). Thus, about one-half of the opening is now lined with new mucosa.

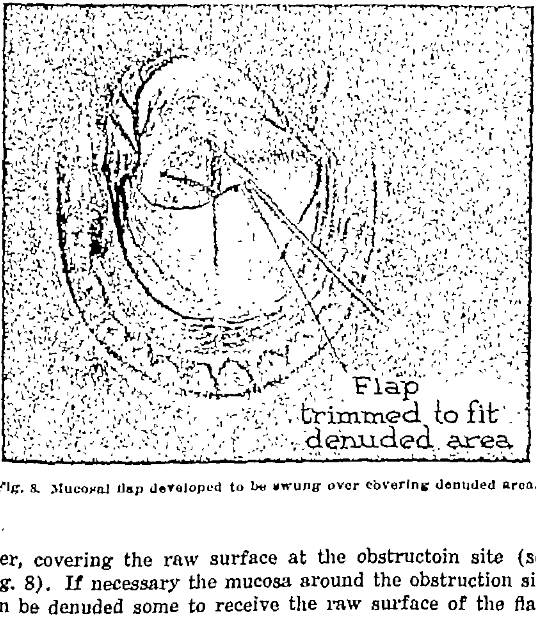

Fig. 8. Mucosal flap developed to be swung over covering denuded area.

All bleeding is controlled and then the palatine mucosal flap is replaced with interrupted 000 silk sutures, working from the posterior poles forward (see Fig. 10). Slight elevation of the adherent anterior margins of the incision facilitates suturing.

No postoperative packing or tubing is necessary and sutures can be removed from the palate the fourth postoperative day.

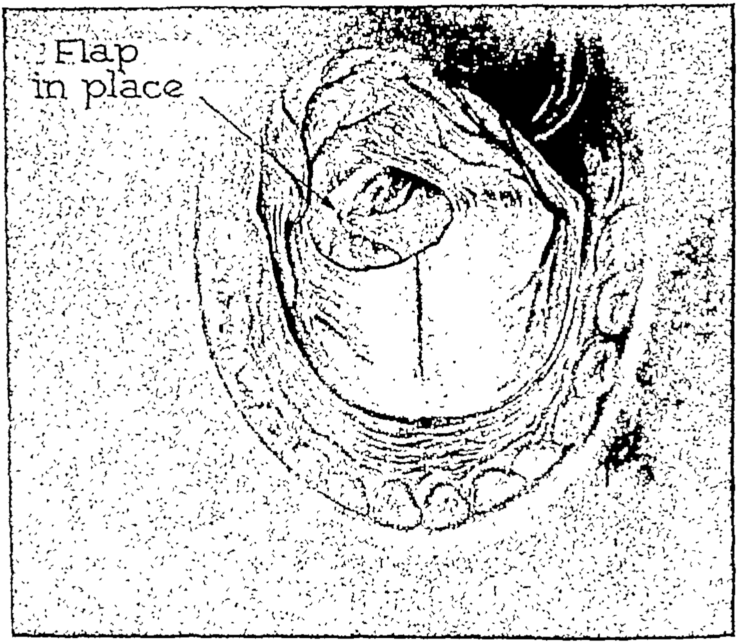

Fig. 9. Septal mucosa flap sutured in place.

Postoperative care consists of keeping the opening patent and free from crusts. The patency is tested by passing suitable dilators.

Dolowitz and Honey[6] described an ingenious way of handling a unilateral bony atresia by a palatine trephine with a submucous resection of the bony obstruction and then handling the mucosal flaps intranasally. The case of bony atresia which I treated was bilateral and needed to be treated in infancy. I feel that it would be difficult to handle any flaps at this age by the intranasal route. It would seem to me that a membranous atresia would also offer difficulty in splitting it to produce a raw surface on the flaps. I am not convinced that flaps help physiologically, because I wonder if correct ciliary action is maintained when we maneuver flaps in different directions or if there are cilia on the surfaces of the obstructing web.

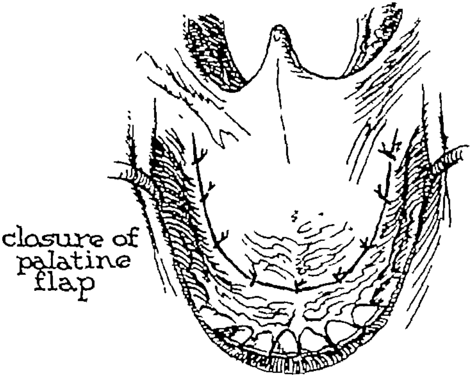

FIG. 10. Palatine flap replaced with interrupted silk sutures.

McGovern observed material passed into the pharynx on the obstructed side after surgery without using mucosal flaps. This was confirmed by Wright *et al.* When flaps are used, there is no doubt that it minimizes cicatricial contraction.

It is interesting to note that Libensohn[10] states that 90 per cent of such obstructions are osseous. Five of seven cases, or 71.5 per cent, in my series were osseous.

My youngest case was, of course, the bilateral bony atresia which I did on a patient six months of age, after first doing intranasal punctures at three weeks of age. My oldest case was 21 years. In all cases the same deformity of crowding of the lateral nasal and lateroposterior epipharyngeal walls of the choana along with atrophy of turbinates was encountered.

The transpalatine approach to the correction of choanal atresia is a very simple procedure that no one with good surgical training should be afraid to perform, and I assure you the results will be most gratifying.

CONCLUSIONS.

1. The exact etiology of choanal atresia is not known.
2. Difficulty in treatment arises because of cicatricial contraction after surgery. This is due to a crowding of the lateral nasal walls and also the lateroposterior wall of the pharynx.
3. A transpalatine approach offers the best means of surgical correction.
4. Surgical correction early with restoration of normal nasal physiology might prevent the poor development of the nasal and pharyngeal cavities on the obstructed side.

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conscious of a stuffiness in the nose, sometimes accompanied by a watery or serosanguinous discharge. A superficial ulceration of the septal mucosa, thought to be on a traumatic basis, may appear and disappear. On the other hand, the initial expression of the disease may be a superficial ulcer in the roof of the mouth or in the gingivobuccal groove. Some patients seem to have had infection in the paranasal sinuses preceding the onset of the lethal granuloma; in others a history or findings suggestive of an involvement of these structures cannot be obtained.

The second stage Stewart[1] termed the stage of active disease. The patient's nasal obstruction may be associated with a small, brownish ulcer on the septum, inferior concha or roof of the mouth, covered with a gelatinous crust which on removal shows a shallow ulcerating surface. The discharge is purulent or sanguinopurulent and has a foul smell. The disease tends to spread from the inside of the nose to the palate, or from the inside of the nose to the outside, so that early in the active stage the tip of the nose may become swollen and indurated. A perforation of the hard palate may be one of the early signs. The disease progresses and sequestra of the nasal bones, the malar bones and the palatal bones may come away. Abscess formation may take place under the cheek and may require drainage. Secondary granulomas may appear on the posterior pharyngeal wall, the nasopharynx or the larynx. There may be a little fever with an irregular temperature and very moderate leucocytosis or even leucopenia. The polymorphonuclear leucocytes tend to maintain their normal proportions in the blood picture. Agranulocystosis has never been found and septicemia as indicated by the blood picture is absent. Except for rather pronounced weakness and lassitude, the patient may present a curious impression of well-being in spite of the serious appearance of the advancing lesion.

In the third or terminal stage, Stewart[1] stated that the obvious exhaustion of the patient is an outstanding clinical feature. The facial appearance may be monstrous. The eyelids are swollen and purulent discharge trickles from between the lids. Sloughing areas appear over the lacrimal sacs and cheeks and the soft tissues about the external nose disappear, perhaps including the upper lip. The mutilating destruction may be extreme. The hard and soft palates may be completely destroyed. Through the large central aperture in the face, the tongue, the roof of the nose and the nasopharynx may be plainly visible, together with the roofs of the maxillary air sinuses exposed by ulcerative destruction of the lateral walls of the nose. The posterior pharyngeal wall may be covered with dirty, necrotic material. The tongue remains unaffected and sequestration of the basisphenoid has never been observed. The patient may die of hemorrhage from ulceration of large vessels which are troublesome to control in a necrotic region, the bony walls of which have been destroyed; however, usually he seems to die from simple exhaustion.

Stewart[1] summarized his clinical description by stating that the picture is one of progressive destruction of nose, face and pharynx. The duration of the illness may be from a few months to five years or more. The most striking feature of the disease is the apparently complete absence of resistance on the part of the patient to the progress of the disease.

Stewart's[1] brilliant clinical description of this disorder can hardly be improved upon.

Weinberg[2] in discussing idiopathic granulomas added what appears to be an important point. He found that these lesions were not strictly limited to the midline tissues of the face, but that here and there throughout the body a pathologic picture of periarteritis nodosa developed.

Teilum[3] stated that, by looking on these lesions from a common pathogenetic point of view, based on morphologic observations and experimental studies on the vascular and tissue changes in allergy, essential support has been obtained for the concept that these lesions are primarily vascular and are of allergic nature.

According to Klinge,[4] the essential morphologic substrate of these related conditions is a swelling of the ground vessels which are involved in the process, with edema, fibrinoid degeneration and swelling of the collagen fibrils in all the layers of the walls of the vessels as the first morphologic change. Klinge believed nonspecific granuloma to be an allergic lesion which pathogenetically is closely related to rheumatic fever as well as to allergic vascular lesions such as periarteritis nodosa and lupus erythematosus disseminatus.

Evidence[5] has been presented which indicates that underlying all the physiologic mechanisms of the body for resistance to stress lies a stereotyped reaction of the peripheral vascular bed. This consists of arteriolar constriction with dilatation which may amount to atony of the conjoined capillary and venule. This reaction accompanies antigen-antibody allergy, but it is a more primitive mechanism and may itself produce the clinical manifestations of allergy without the intervention of an antigen-antibody reaction.

Kahn[6] has suggested that allergy is merely an expression of hyperimmunity and that there are many examples in physiology of the overactivity of a physiologic mechanism.

Walsh, Sullivan and Cannon[7] have demonstrated that immunity may be localized to or be greater in certain tissues than others.

The midline tissues of the face is a region where skin and subcutaneous tissues and mucosa and submucosal tissue come in close relationship. These tissues are among those most capable of developing an intense immunity and consequently a hyperimmunity, and consequently it is not unreasonable to suppose that they might readily become the site of a destructive granulomatous reaction of an allergic nature.

Differential Diagnosis: From the purely clinical standpoint it seems apparent that there is nothing in the gross picture of this condition to distinguish it from specific infectious granulomas occurring in the same locations except the appearance elsewhere in the body of lesions of periarteritis nodosa or lupus erythematosus disseminatus or rheumatism.

Pathologists are careful to report sections taken from these lethal granulomas as being not "typical" or "diagnostic" of this or that condition, being prepared to admit that an atypical example of any one of the specific granulomas might be present.

Hoover[8] gave a typical pathologic description of one of these idiopathic lethal granulomas. He stated, "On the border of the lesion is an amorphous mass of necrosis beneath which there is an infiltration by inflammatory cells, chiefly lymphocytes and macrophages with occasional plasma cells. A few polymorphonuclear leucocytes are also present. The large vessels in the tissue show a definite perivascular cuff of lymphocytes and macrophages with degeneration of arterial walls in some regions. While there are focal accumulations of infiltrating cells in the tissues, the lesion is not characteristic of tuberculosis, syphilis or leprosy." The pathologist added that the lesion suggested the Arthus phenomenon.

Weinberg[2] pointed out that oidiomycosis, glanders, tularemia, leprosy, rhinoscleroma, blastomycosis and sporotrichosis all give a somewhat similar histologic picture and attempts to demonstrate these organisms as etiologic agents should be made. He also considered the possibility that a virus might be the causative agent, but stated that of the viruses, only that of venereal lymphogranuloma produces a microscopic picture which is similar.

Weinberg believed that if these organisms are eliminated insofar as possible, there is left a picture which clinically consists of ulcerations of the upper respiratory tract associated with evidence of focal alterations in the lungs, and which anatomically shows multiple granulomas of unknown origin involving one or more organs with terminal development of periarteritis nodosa. He believed that the constancy of the history of antecedent chronic infection in these cases lends support to the belief that hypersensitivity may be a factor in their development.

The evidence presented seems convincing that idiopathic lethal granuloma of the midline facial tissues is an instance of localized hyperimmunity resulting in an Arthus-like reaction of the involved tissues.

REPORT OF CASE

The patient is a woman 50 years of age In November of 1919 the Eustachian Tubes were inflated because of a pounding in her ear There was some trauma on the right side followed by pain in the nose which gradually grew worse A week later she observed an ulcer on the right side of the septum. About two weeks later or around Dec 1, 1949, she developed a blood tinged, yellowish discharge from the right nostril associated with burning and tenderness of the nostril. Swelling of the tip of the nose followed, but did not persist The discharge continued. The process soon extended to the left side and crusting and epistaxis developed Treatment consisted of the local use of sulfathiazole ointment and oil nose drops and the systemic use of aureomycin, penicillin and one of the sulfonamides

She was referred to the Mayo Clinic on Jan. 7 1950 An ulceration in the anterior portion of the septum extending backward about 2 cm., covered with granulation tissue was found The rest of the nasal mucosa appeared normal Sinus X rays showed a slightly thickened membrane in both antra. There was a secondary anemia, but the differential leucocyte count was normal and blood smears showed nothing of significance

A biopsy was taken from the nose and sent to the Sections on Pathology and Clinical Bacteriology of the Mayo Clinic. The pathologist reported the tissue to be a granuloma with foreign body giant cells and eosinophiles. There was considerable subepithelial fibrosis. Bacteriologically, the material was studied extensively in various special media, both aerobically and anaerobically Micrococcus pyogenes and streptococci fecalis were found Repeated cultures for tuberculosis, Brucella and fungi were negative. They were unable to isolate a virus. There were three negative guinea pig inoculations Kline, Kahn, Hinton and Wassermann tests for syphilis were negative A diagnosis of idiopathic lethal granuloma of the midline facial tissues was made

Comment Because the evidence from the literature suggested that this condition was an allergy, nonspecific and antiallergic treatment with the vasodilator, monoethanolamino nicotinate (Nicamin Abbott) was given. The patient was dismissed to the care of her home physicians.

The patient seemed to show improvement after first returning home, but in March she developed hoarseness which had grown worse, and this was accompanied by an increasing dyspnea. The ulcerating granuloma was then seen to involve both sides of the septum and the anterior ends of the inferior turbinates. The interior of the nasal cavities could not be inspected because gentle attempts to cleanse the nose of crusts and mucopus produced bleeding There was a pale granulomatous mass in the nasopharynx. There was granulomatous involvement of the anterior half of the left vocal cord and a smaller granuloma on the right arytenoid. Chest X rays were negative. She complained of loss of appetite, strength and about 20 pounds of weight. There had been shooting pains in her legs for the previous two weeks. Nicamin was continued and terramycin, 250 mg. every four hours, was added

From May 5 to June 29 she was on intermittent treatment with terramycin. By May 25 the nasal picture had improved, but the larynx had become more involved and there were large subglottic granulomata. Breathing was embarrassed to the point where a tracheotomy was considered. During the rest of this period, however, the nasal granulomata became firm and showed less tendency to bleed. There was regression of the subglottic granulomata with improvement in breathing, but none in her voice. This was probably the result of the effect of the terramycin on the secondary infection rather than on the primary process.

On June 28, 1950, cortisone was obtained for her and she entered St. Luke's Hospital, Cleveland, Ohio, for further study and treatment. The patient refused to allow a biopsy from her larynx or repetition of one from her nose. After much persuasion a small biopsy was taken from the nose on July 14 and used for special cultures with the same negative results which had been obtained at the Mayo Clinic.

On admission to the hospital, the following interesting family history was obtained. One sister had leg ulcers with involvement of eyes and lungs which was suspected to be tuberculosis. This is now arrested, but she had many contacts with the patient. One half-brother and a niece had been in a sanitorium with suspected tuberculosis. No definite diagnoses had been established. The patient was found to have papulopustular lesions on the elbows and right foot. Blood chemistry was normal; the glucose tolerance test was normal. She still showed a secondary anemia and there were 10 per cent eosinophiles in the differential count. An intravenous epinephrine test of the pituitary-adrenal mechanism was given. The circulating eosinophile count at 7 A.M. before the epinephrine was 346 cmm. At 10 A.M. this count had fallen to 229 cmm., and at 11 A.M. to 170 cmm., which was considered a normal response.

On July 10 one of the papules was removed from the elbow. This was examined by Dr. Raphael Dominguez and Dr. A. J. Segal. They reported necrosis of the epithelium in the central portion with the necrosis extending beneath intact epithelium on one side. There was a heavy infiltration of polymorphonuclear leucocytes and round cells with no evidence of malignancy. Stains for bacteria and fungi were noncontributory. Their diagnosis was necrotizing granulomatous dermatitis, cause undetermined. Slides of this tissue were sent to the Mayo Clinic where they were examined by Dr. A. C. Broders, who thought that it was a Grade I hemangioendothelioma representing a Kaposi tumor. He reviewed the tissue that had been removed at previous biopsies and agreed that this diagnosis could not be made from these slides. The patient had other lesions on the elbows, thumbs, fingers and feet which were identical in appearance to the one removed from the left elbow for biopsy. On her return to the Mayo Clinic these lesions were observed by a consultant in the Section on Dermatology and it was his opinion that these lesions did not resemble Kaposi's tumor.

The clinical course of this patient, while bearing some slight resemblance to that of Kaposi's tumor, is much more characteristic of that of idiopathic lethal granuloma; moreover, Dr. Frank B. Hazard, of Cleveland, and Dr. Joseph C. Ohlmacher, of Vermillion, S. Dak., pathologists to whom these tissues have been submitted, have not been convinced that the area in question was truly representative of an endothelioma, but have thought it probably representative of the necrotizing vasculitis seen in both specific and nonspecific granulomas. It was, therefore, felt that the weight of the evidence was on the side of the lesion being an idiopathic granuloma and that treatment should be given on this basis. Since idiopathic granuloma was thought to be due to a local hypersensitivity with the stereotyped reaction of the peripheral vascular bed seen in these conditions, the use of cortisone was advised.

Cortisone in Allergic Reactions: Most of the conditions in which cortisone has been found effective have been the disorders which are usually referred to as the "collagen diseases." The evidence suggests[7] that the term "collagen disease" is synonymous with allergy; in other words, localized areas of tissue hypersensitivity. Among the disorders in which the local lesions have responded favorably to cortisone therapy are rheumatoid arthritis, vasomotor rhinitis, asthma, periarteritis nodosa, lupus erythematosus disseminata and certain diseases of the eye, all of which are grouped under the collagen diseases and, therefore, may be reasonably presumed to be allergic. The exact manner in which cortisone influences the functional and histologic lesions in the allergies has not been worked out.[10] Since the evidence suggests that lethal granuloma is also a collagen disease (or an allergy) it was decided that cortisone would be worth a trial in this condition. One of us (Williams[6]) had previously reported a case in which cortisone had apparently had some favorable effect.

The patient accordingly was placed on 200 mg. of cortisone a day for two days and then 100 mg. a day. Four days later she was feeling much stronger and had less general aching. Her breathing was improved and there was less drainage from her nose. The sedimentation rate was 41 mm. per hour. The tenderness of the skin and joint lesions had disappeared.

We were concerned over the possible effect of cortisone on the healing of the site of the biopsy. This, however, healed normally and the sutures were removed on the fourth day.

Nine days after starting cortisone the glucose tolerance was decreased and she was put on a diabetic diet and iron. The sedimentation test was approaching normal and the circulating eosinophile count was within normal limits.

On July 21 she was discharged from the hospital on 50 mg. of cortisone a day, ferrous sulphate and potassium iodide. At that time the turbinates appeared atrophic and there was a great deal of nasal crusting. The vocal cords were still red, but the granuloma on the left cord was barely visible. A week later there was a definite depression of the bridge of the nose and a large perforation could be seen in the septum. The granuloma appeared to be markedly decreased and dense scar tissue was apparent in the area. The voice gradually increased in strength and her general strength and weight improved. This patient, however, remained on 50 mg. daily for 41 days and while there was no further improvement in her condition, neither did it regress, the *status quo* being maintained.

Cortisone was stopped on September 2, and three days later pain and swelling of both ankles returned. She returned to the Mayo Clinic and by Sept. 11 she was having severe dyspnea from an increase in the subglottic granulomata. She was seen by a consultant in the Section on Rheumatology and a diagnosis of rheumatoid (atrophic) arthritis of the knees and ankles was made.

There had been a very marked change in the lesion in the anterior portion of the nose since she was last seen. The granuloma in this area had entirely disappeared and was replaced by dense fibrous tissue which had narrowed and deformed both nasal vestibules. The bridge of the nose in the area between the upper and lower lateral cartilages was pulled in. A large septal perforation was present. The area in the nose resembled the scarring of rhinoscleroma. There was a granulomatous mass in the nasopharynx.

Inspection of the larynx showed diffuse involvement of both vocal cords, but the major involvement was in the subglottic region. A tomogram taken of the laryngeal region was reported to reveal a large, soft tissue mass in the region of the thyroid and cricoid cartilages which was producing lateral compression from both sides in the subglottic region. The patient was seen by a consultant in the Section on Laryngology who advised emergency tracheotomy. Because of the nature of the involvement and the previous good response to cortisone, it was considered wiser again to try the effect of this hormone.

The skin lesion on the right foot had also enlarged and ulcerated. The dermatologic consultant thought that this lesion might represent a necrotic dermatitis of the type seen in lupus erythematosus disseminatus.

The Cortisone Committee of the Mayo Clinic allotted enough cortisone for three weeks' treatment.

The patient was placed on 200 mg. of cortisone daily for four days. This was followed by 100 mg. a day until the end of the three weeks' treatment period, Sept. 26. The pain and stiffness of the knees and ankles had entirely receded by the end of the third day of treatment, the ulceration on the right foot had nearly disappeared, the patient's dyspnea was entirely relieved, and her voice was more normal. Another tomogram of the laryngeal region taken at this time was reported to show a 50 per cent reduction in the size of the soft tissue mass with disappearance of the encroachment on the airway.

The patient was again dismissed to the care of her home physicians; but three days later, due to a recurrence of the arthritic pains in the knees and ankles, the cortisone had to be resumed. It was again stopped on Nov. 25.

During the first two weeks of December the patient had abdominal pains, diagnosed as being due to a pancreatitis with calcification. She also had some blood in her urine, and tests showed the kidney function to be decreased. Except for these episodes she continued to improve and 18 days after she had discontinued cortisone the vocal cords looked relatively normal and the subglottic granulomata had almost disappeared; however, at the end of four weeks she again became dyspneic and the subglottic granuloma was large and red. She was again given cortisone.

Comment: By Dec. 28 she had received about 9.5 gm. of cortisone. Except for its effect on her glucose tolerance and some possible kidney involvement, she has shown no ill effects from its continued use. When the cortisone has been stopped, the granulomata have increased in size rather promptly, but the last period before relapse was about four weeks as against only a few days previously; however, at no time has she been completely free of her lesions except for the vocal cords and the skin.

During the course of her illness she has shown granulomata of the nose, nasopharynx, larynx, trachea and skin of the elbows, thumbs, fingers and feet. She has also shown some episclerotic injection of the eyes. At one time while at home she had a red, painful swelling of the left auricle which subsided without further involvement. We feel that the most encouraging feature in this case is the replacement of the initial lesion of the disease in the anterior portion of the nasal cavities by scar tissue. We hope that continued use of cortisone will encourage such changes in the granulomatous lesions in the nasopharynx and larynx.

Summary and Conclusions: A 50-year-old woman developed an idiopathic lethal granuloma of the nose which secondarily involved the nasopharyngeal and laryngeal regions. A rheumatoid arthritis of the knees and ankles developed synchronously with the laryngeal involvement, and lesions resembling the skin lesions of lupus erythematosus disseminatus developed on the elbows, hands and feet.

The results of cortisone therapy appear to encourage further trial of this hormone in this previously lethal disease.

At this time (March 15, 1951) there is no visible granuloma in the nose, nasopharynx, larynx or trachea. The skin lesions have disappeared. This has been the case since Feb. 20, 1951. On Feb. 18, 1951, she was switched to ACTH, 25 mg. every six hours. This was reduced on Feb. 21, 1951, to 10 mg. four times daily. She still has some abdominal discomfort, nausea, blood and albumin in the urine and generalized edema. Since being put on ACTH she has developed marked pigmentation of the skin typical of that seen in Addison's disease. There is no pigmentation of the mucous membranes.

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INTRALARYNGEAL ARYTENOIDECTOMY WITH OBSERVATIONS IN THREE CASES.*

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I appreciate this opportunity of appearing on the program, and I should like to speak on the subject of intralaryngeal arytenoidectomy and present some of my observations concerning three cases.

Case 1: Forty-five-year-old Mrs. E. D. became known to us as "Calamity Jane." An an adopted child without any knowledge of her family, she did very well until the age of 19 when she had an appendectomy, and 19 years later she had a kidney operation which was followed by partial right hemiplegia from which she recovered. The next year brought an operation for adhesions, and three years later there was incised and drained a rectal abscess. This brings us up to 1946. Shortly after the rectal abscess, she developed signs and symptoms of thyrotoxicosis and enlargement of the thyroid gland. On Feb. 23, 1947, my examination of her larynx gave essentially normal results in all respects. I did not see her again until April, 1947, but the story goes that on Feb. 24, 1947, she was operated upon for thyroidectomy and during the course of the operation she developed dyspnea, for which a tracheotomy was necessary. Microscopically, tissue from the thyroid gland proved to be malignant, and during the weeks following the operation the malignant tissue grew out into her neck and exuded through the thyroid incision. Ninety-nine hundred Roentgens of X-ray therapy produced astoundingly gratifying results. On April 16, 1947, I saw this patient again, and at that time there was complete paralysis of the right vocal cord; but there was a little movement of the left vocal cord. She apparently had adequate breathing space, the tracheal tube was removed on April 20, 1947, and she was allowed to go home. Her voice was poor.

Seven months later, using local anesthesia, a tumor was removed from one arm, and this proved to be benign. During the course of the next year she developed pain in both arms and in December, 1948, under pentothal sodium anesthesia, cervical laminectomy was performed for relief of the pain. During the course of the operation, she developed acute dyspnea, and a tracheal tube had to be inserted. This tube was removed about two months after the operation.

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During the months that followed this last operation, she noticed that her throat had become more sensitive, and that she had noisy inspiration. There were attacks of laryngeal spasm Her friends told her that she snored loudly at night. She was examined in my office some months after the last operation, and being unaware of her recently developed sensitive throat, I inadvertently set off an attack of laryngeal spasm by placing a laryngeal mirror in the oropharynx. The spasm lasted for about 15 minutes, and caused cyanosis and considerable fright to the patient. Attempted examination in the hospital under mild sedation was followed by another attack of laryngeal spasm which was of longer duration than the office attack and necessitated the administration of oxygen Because of the increasing dyspnea and the attacks of laryngeal spasm, a size 5 tracheal tube was reinserted with complete respiratory relief Obviously she had not had adequate airway By laryngeal examination it could be seen that the right vocal cord remained immobile, and there was the same slight movement of the left vocal cord

On Nov. 7, 1949, she was operated upon for arytenoidectomy by the method to be described Two weeks later the tracheal tube was removed, and in approximately four weeks from the time of operation she was dismissed from the hospital

Since the arytenoidectomy, she has not had respiratory difficulty, and there has not been an attack of laryngeal spasm, even when the larynx has been indirectly examined, moreover, she has not had any operation during these 14 months Her voice remains much the same as it has been since the original thyroid operation

Case 2 is that of 55-year-old Mrs A. G, who was operated upon for thyroidectomy on Sept. 27, 1949 She was unable to talk when she awoke from the anesthesia, but there was no dyspnea. For three months her voice improved, but then she began to have occasional "strange, loud and heavy" noises to erupt spontaneously from her throat, and there was shortness of breath on exertion Six months after the operation she suffered a severe attack of dyspnea, which was followed in three days by a second such attack, because of which a tracheotomy was done by her general surgeon and the anesthetist. Mirror examination showed that she had bilateral abductor paralysis of the vocal cords Right arytenoidectomy by the method to be described was performed one week after the tracheotomy. Her tube was stoppered in two weeks and removed in four. By mirror examination, it seemed that she had an excellent result, and she felt that she had adequate breathing space. Her voice was good. Seven months later, however, she came to my office for an interval visit, and there she stated that she was breathing and talking all right, but she was so tired all the time that she felt that she could not live. She was most tired when she awoke in the mornings. Inquiry of her neighbors brought out the fact, completely unknown to her, that her breathing at night was so loud that it disturbed them. Those persons living in apartments closed to the patient were very desirous of moving elsewhere Three days later I reinserted the tracheal tube, and on the following day she told me that she awoke from the anesthesia "rested." She went back to work in a few days, and she said that she felt so well that on her first day back in the office she caught up on the work that had accumulated during her absence Obviously her operation for arytenoidectomy to relieve respiratory obstruction due to bilateral abductor paralysis thus far has failed. Further operation is contemplated.

Case 3 is that of a 38-year-old colored farmer. His story began with a gunshot wound of the throat while he was in the army in 1942. From membrane is dissected from the arytenoid cartilage. As soon as convenient (see Fig. 3) the arytenoid cartilage is grasped with laryngeal alligator forceps, and the dissection is completed with scissors and knife. After the cartilage is removed

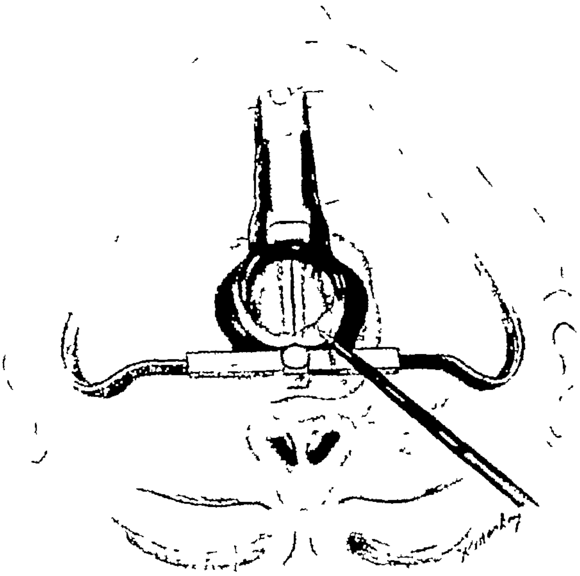

Fig 2 Exposure of the larynx by means of the Lynch suspension apparatus Mucous membrane over the condemned arytenoid cartilage is injected with monocaine hydrochloride solution

hemostasis is accomplished by the use of the electrocoagulation current through the laryngeal length electrode. Finally (see Fig. 4), a mattress suture of 00 chromatic catgut is placed so that it enters the lateral aspect of the aryepiglottic fold, passes through the posterior end of the vocal cord, and is passed back through the aryepiglottic fold close to its point of entry. It is not the purpose of this suture to close the incision; it serves only to pull the cord laterally. The incision is not sutured. This provides adequate drainage for serum and blood.

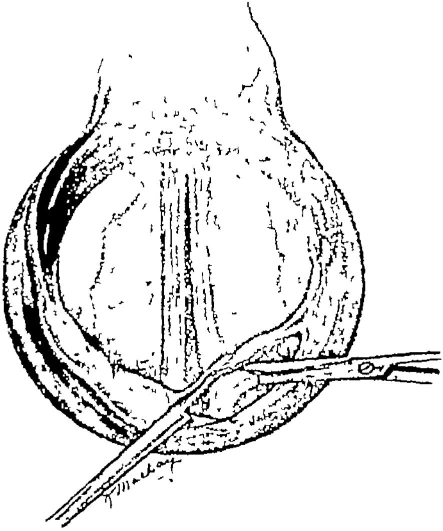

Fig 3. Removal of the arytenoid cartilage.

Postoperatively for the next three days the diet is liquid, partly because the patient's throat is sore, and partly because it facilitates a cleaner throat. The patient may be up as soon as the effects of the anesthesia have subsided. The tracheal tube is stoppered at about 10 days, and removed in two to three weeks, according to the results of the mirror examination, and the patient's symptoms.

There are several advantages to this method: *a.* Simplicity. The operation is less formidable, as a rule it is less time consuming, and the arytenoid cartilage is so much more easily located. *b.* The possibility of wound infection is reduced. *c.* There is less likelihood of the postoperative formation of hematoma which may cause a reduction in the calculated

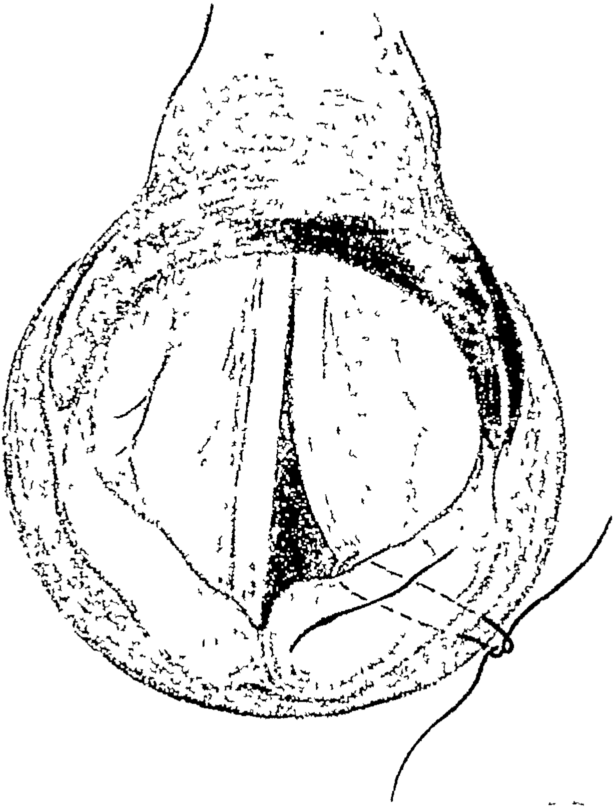

Fig 4 Placement of the mattress suture to laterally displace the vocal cord.

width of the glottis. *d.* When working directly in the larynx, the surgeon has immediate and constant view of the amount of space that is being created between the vocal cords.

There are only two disadvantages: *a.* Rarely there may be technical difficulties in the use of the Lynch suspension apparatus. *b.* There arises the question of whether the vocal cord can be displaced far enough laterally without suturing it to the wing of the thyroid cartilage. Only statistics will answer.

As to when the patient should be operated upon for bilateral abductor paralysis of the larynx, the consensus of opinion is that if the paralysis remains total six months postoperatively it can be considered permanent.

For the results in these three cases let us consider that there are three classifications of results: the *good* without reservation, the *good* with reservation, and the *poor.* By the second classification I refer to those cases which fulfill some of the criteria for a successful result, but do not fulfill all of the criteria. They may be able to get along, but they have not been returned to the preoperative calculated rehabilitated state. Classes 1 and 3 are self explanatory.

From this classification there might be some individual opinion as to the results. At worst, the batting average would be 33 per cent and at best, 66 per cent. I rather incline toward the latter, since in my opinion, the patient described in Case 1 has been rehabilitated, and so far as we know, the patient in Case 3 has been rehabilitated. Case 2, at the time of this writing, provides us a failure.

I should like to emphasize that in considering the treatment of bilateral abductor paralysis of the larynx, it is necessary to consider many things: the size and shape of the larynx, the sex of the individual, the patient's psychology. The result that could be called a success for the person who does sedentary work might be called a failure for the laborer. The result that could be called a success for the person who does not have to use his voice except to make his wants known might be a failure in a clergyman or a teacher. If time should prove that similar results can be obtained by the extralaryngeal and the intralaryngeal approaches, the latter will surely have one thing in its favor: ease and simplicity of procedure.

In conclusion, I pay my respects to some of the workers in the field of the treatment of the symptoms of bilateral abductor paralysis of the larynx, Hoover,[1] Loré,[2] King,[3] Kelly,[4] and two members of our present company, Dr. Clerf[5] and Dr. Jackson,[6] but to William C. Thornell,[7,8] of Cincinnati, duly goes the credit for first having brought to the surgeons' attention the intralaryngeal approach to arytenoidectomy.

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137 Doctors Building.

OSTEOMATA OF THE FRONTAL SINUS WITH SPECIAL CONSIDERATION OF THE SURGICAL REMOVAL.*†

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When the literature regarding this essentially or histologically benign group of tumors is reviewed, one is surprised at the various moot questions that are presented. Among them are: *1.* pathogenesis; *2.* base of origin or attachment; *3.* frequency of incidence; *4.* therapeutic indications; and *5.* possibly, the surgical procedures and technique to be followed.

Pathogenesis: Carmody[1] makes clear the controversial opinions, as to etiology, that have been presented, naming four theories:

1. That they arise from embryonic cartilaginous cells at the junction of the ethmoidal and frontal bones.[2] (Arnold.)
2. That the origin is from the periosteum of the frontal sinus walls.[2,3] (Fettisoff.)
3. That the diploe is the generating base.[3] (Virchow.)
4. That some inflammatory stimulus is the underlying reason, suggesting the possibility of lues, tuberculosis, trauma,[3] (Cushing) or sinusitis.[4] (Eagleton.)
5. Another is added by Cloquet[3] who believed them to be ossified polyps.

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On the other hand, Leeds[5] believes that no completely satisfactory explanation has yet been submitted.

Kelemen,[6] in one of his four reported cases, saw histologically an area that reminded him of otosclerotic bone changes.

He also believes that, as a rule, the symptoms are expected to disappear after proper radical surgery.

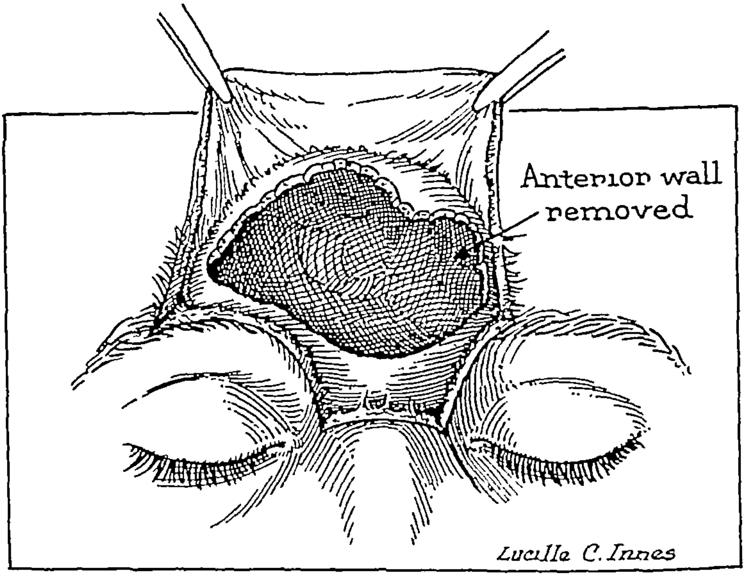

Fig. 1. Case 1. M. J. Removal of anterior plate by gouge and rongeur. There was more bone removed than shown, including some of the left anterior plate and the right supraorbital ridge, making for postoperative deformity.

Austin J. Smith[7] feels that there is a question as to whether the osteomata are true tumors. He states that pathologically (histologically) they are related to the so-called fibrocystic group of bone lesions.

H. Brunner and Spiesman[8] point out that there are two types of expansion of osteomata: some expand by increase in the size of the tumor; others expand by the formation of mucoceles. (Noted within the orbit in this current Case 1.)

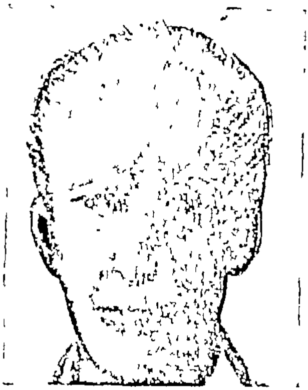

Fig. 2. Case 1. M. J. Photograph taken a few years after surgery, showing the marked depression

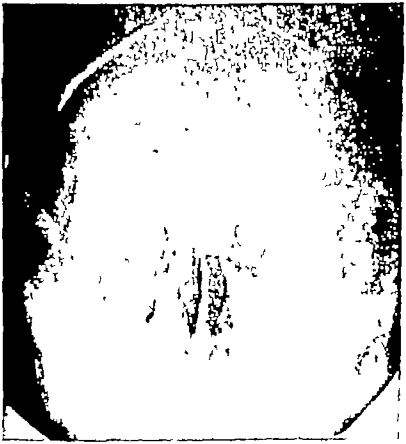

Fig. 3. Case 2. E. J Preoperative X-ray film showing large tumor.

The increase in the tumor size is due to a metaplastic ossification of the connective tissue and to a deposition of new bone by the paranasal mucosa which serves as periosteum. The tumors thus consist of a spongiose core, and a solid cortex.

Osteomata of the frontal sinus are divided into two classes histologically:[9]

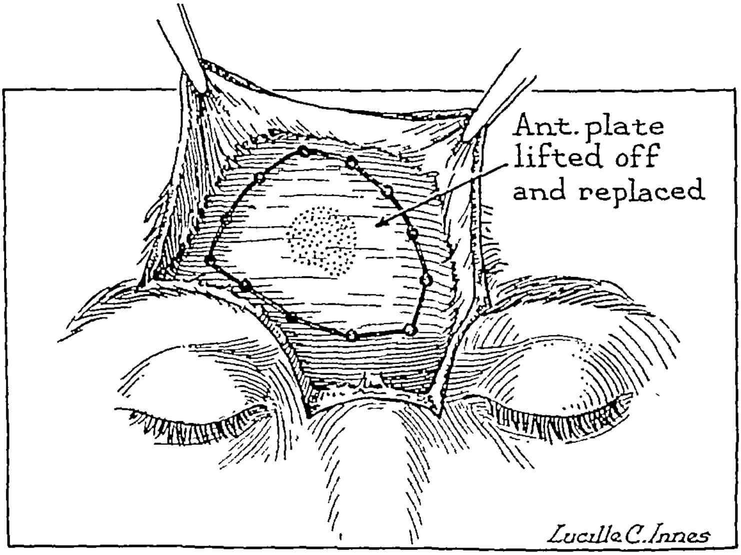

Fig. 4. Case 2. E. J. Lid operation, showing removal and later replacement of the area of anterior plate. The dotted area indicates the bone erosion on the inner surface caused by pressure of the tumor.

1. Hard, dense bone; and *2.* relatively soft, spongy, cancellous bone. There is another classification into two varieties:

1. Pedunculated, and *2.* sessile.

Study of a considerable number of case histories leads one to wonder whether these two methods of classification are not the description of just two varieties: *1.* The pedunculated, hard tumor; *2.* the sessile, soft growth.

Further consideration of the clinical reports brings one to the conclusion that, probably the pedunculated hard tumor is indeed a true neoplasm, springing from a soft generative base, quite likely, and most usually, an embryonal rest. On the other hand, the sessile, soft mass is often described as accompanied or preceded by evident widespread bone changes. Practically all of the frontal area, or even more or less of the skull in general, may reveal X-ray evidence of thickening and softening. It may be that the real etiology is a metabolic bone hypertrophy and that the so-called osteoma in the frontal sinus is only the unbridled thickening, invited by the empty chamber; and in fact is not a neoplasm but a local manifestation of hyperostosis, due to metabolic disorder.

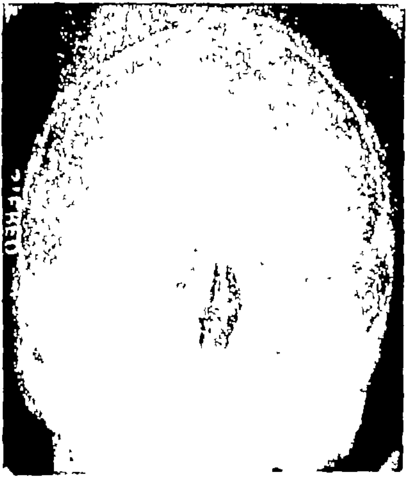

Fig. 5. Case 2. E. J Postoperative X-ray film, after five months.

When the hard tumor is loosened from its bed, the soft generative bone may and should be easily curretted away until the underlying normal appearing bone is reached. When the soft, spongiose growth is surgically attacked, underlying normal appearing bone is *not* reached. If the dissection of the soft bone is followed it leads to the end of all osseous tissue, and one arrives within the cranial cavity, with dural exposure. This may explain the advice of some operators that it is not necessary to remove all of the growth and that the partial removal is not followed by recurrence. Coates describes carefully approaching the depth of the dissected excavation so as to leave a tissue paper thin layer of this bone over the dura, avoiding exposure. When, as in his case, the

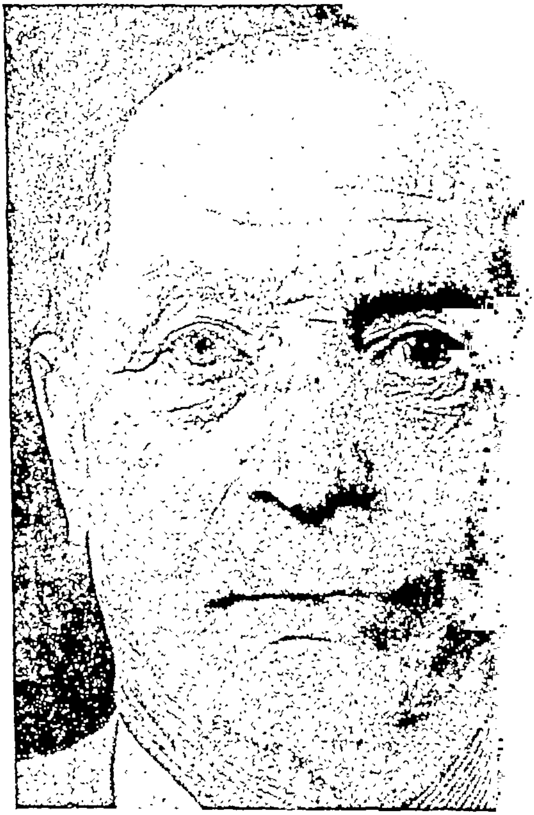

Fig. 6. Case 2. E. J. Postoperative photograph, showing dimple or retracted scar where the area of bone erosion was. Picture taken nearly six months after surgery.

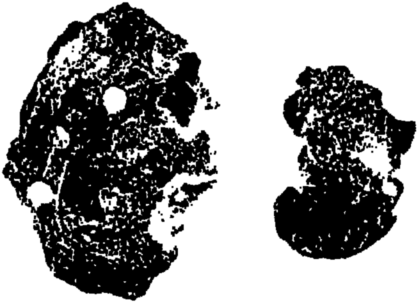

Fig. 7. Case 2. E. J. Osteoma removed in two pieces. Unfortunately, a scale was not placed beside the bone masses. The unbroken mass was approximately 33 mm. broad by 16 mm. deep, and 31 mm. from bottom to top.

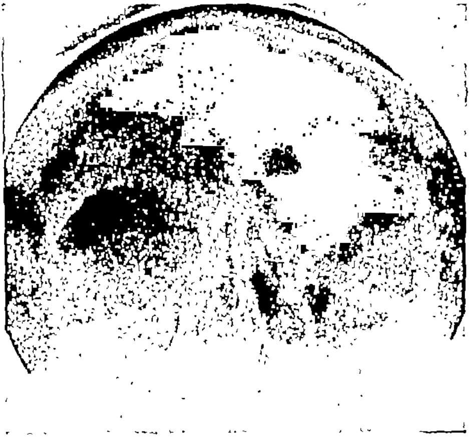

Fig. 8. Case 3. C. B. X-ray film 12 years preoperative.

dura is actually exposed, he advises leaving bone chips over the dura to encourage re-formation of bone.

The Base of Origin: Obviously the variations in the possibilities of base run with the numerous theories of etiology, and many are to be found in the literature.[1,10]

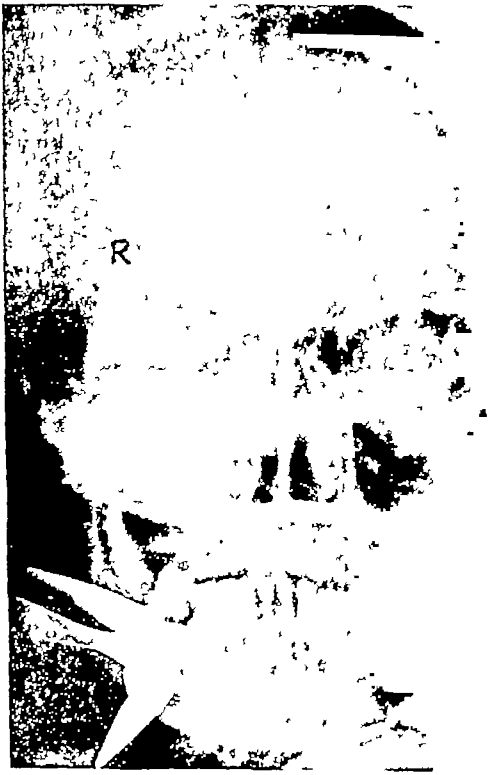

Fig 9 Case 3 C. B X-ray film shortly preoperative, showing marked growth of tumor in the 12 years.

1. Junction of the frontal and ethmoid bones, far forward.
2. The under and inner wall of the frontal bone and the ethmoid capsule.
3. A pedicle inseparable from its frontal bone and the ethmoid origin.

4. Continuous with the frontal bone at the inner wall of the sinus.
5. The upper lateral wall of the frontal sinus.
6. The infundibulum.
7. The floor of the frontal sinus external to the duct.

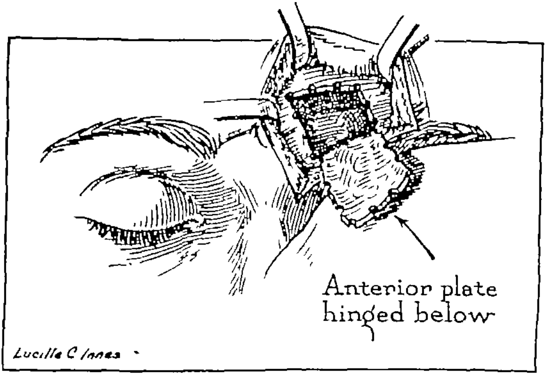

Fig. 10. Case 3. C. H. Hinge operation, showing technique of loosening the anterior plate area on the lateral and superior borders, with the bony area then swung downward over the eye, preserving the soft tissue hinge and thus the blood supply from below.

8. The anterior end of the junction of the ethmoid with the frontal bone.
9. The septum between the frontal sinuses.

Sprowl[3] states that they are usually attached by a cancellous pedicle to the base. In surgical removal, he declares that not only the tumor mass and its pedicle but also the base must be removed to prevent recurrence.

Frequency: The position taken on this question by various observers naturally is largely influenced by their own clinical experience. Certainly the literature does not list any great number of cases, studied and reported.

Dr. Gilbert Owen, who is to discuss this paper, from the standpoint of his observations of referred cases studied by the X-ray, might, reasonably take the position that these tumors are not infrequent. He has seen a series of over 30 cases.

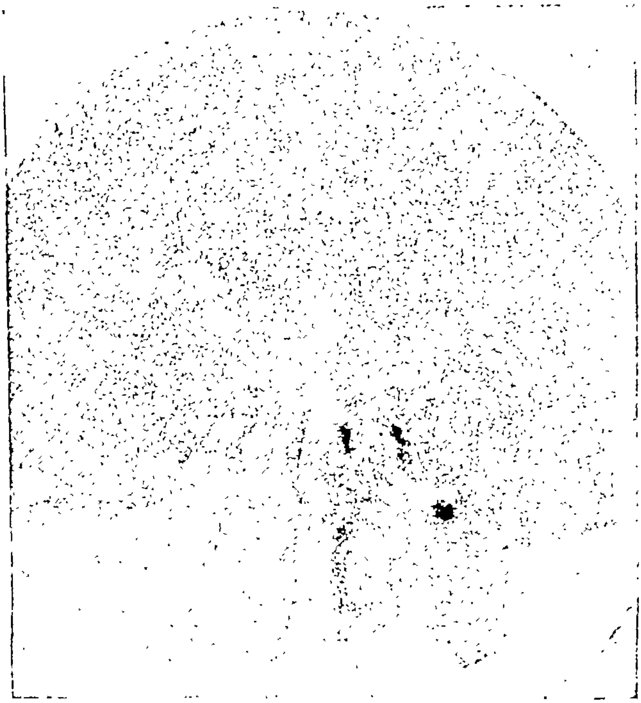

Fig. 11. Case 3. C. B. Postoperative X-ray film showing normal bone texture.

On the contrary, the author found his first case only after 17 years in the specialty. At that time he had never been fortunate enough to have seen a single case operated upon or diagnosed in any clinic, conference, or postgraduate course, here or abroad. His second case did not appear until 22 years later. This experience would tend to lead to the conclusion that such tumors are quite infrequent, if not, indeed, rare.

Carmody,[10] in 1935, collected 139 cases, giving references to the reporting articles, with the author and the journal.

In a series of 3,510 Roentgenograms of the nasal sinuses,[4] osteomata were noted in 0.42 per cent. This seems to have been a rather surprising observation. The first osteoma to have been found by Roentgen examination was reported by *Cappy*[4] *in 1899.*

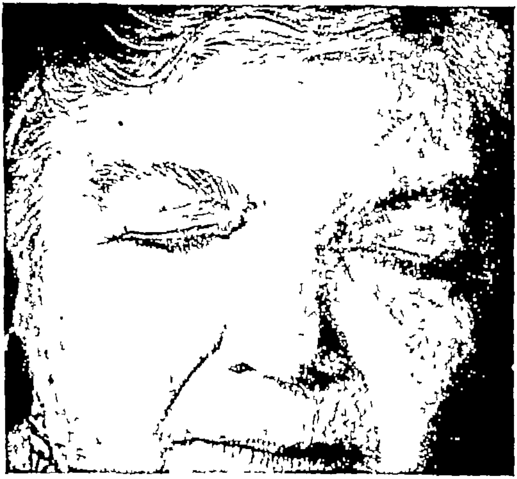

Fig. 12. Case 3. C. B. Postoperative appearance of forehead. As the days followed there was no untoward reaction or swelling

In 1935, Beck stated that in his practice he had had four cases.[10]

In 1936, Sprowl[3] took the position that osteomata of the nasal accessory sinuses were uncommon, the greatest number having been observed in the frontal sinuses.

Reeve,[11] as early as 1918, joins Friedenberg in offering an apology for reporting two cases of his own (one seen in 1881 and the second in 1895) because "of the number of cases already on record." His own excuse was, "That it is desirable to make statistics as correct as possible." Nevertheless, in discussing Reeve's paper, Edward Jackson calls such a tumor a "rare condition." By that time he had had only one case in his own practice, and that some 26 years previous. He had seen two other cases in consultation.

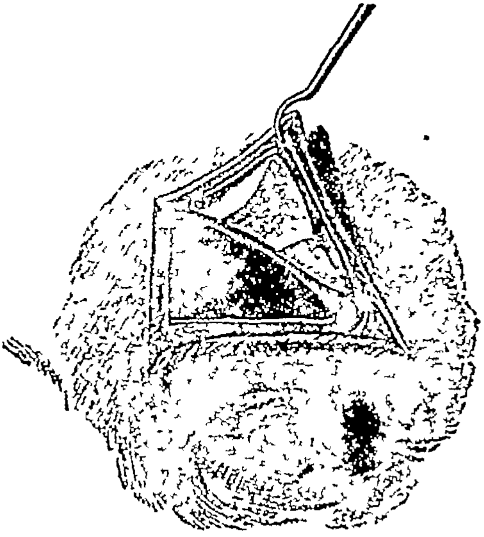

Fig 13 Showing position of the skin-bone flap in osteoplastic resection of the anterior wall of the frontal sinus (Illustration taken from Prof M Hajek's book Pathology and Treatment of Inflammatory Diseases of the Nasal Accessory Sinuses, published by C V Mosby Co, St. Louis, Mo, 1926, and translated into English by Dr J D Heitger and Dr French K Hansel

In that same year, Sewell[12] presented a most interesting article. He considers several of the moot questions discussed in the current paper. He mentions that in 1880, Berlin advised the enucleation of an eye, rather than to try to remove a tumor that had broken through into the orbit. He quotes William MacKenzie as stating, in 1854, that no case of frontal sinus osteoma had appeared in the literature. In discussing the origin as taking place in cartilaginous rests, he does not refute the possibility on occasion, but affirms that this explanation alone cannot be accepted for all such tumors In his paper, in 1918, he reports that his case was the ninety-third to appear in print to that date Among the cases, in the pathology, under the etiology he does not discuss the sessile type of tumor He is most enthusiastic in regard to the Roentgen rays being the chief means of making a diagnosis

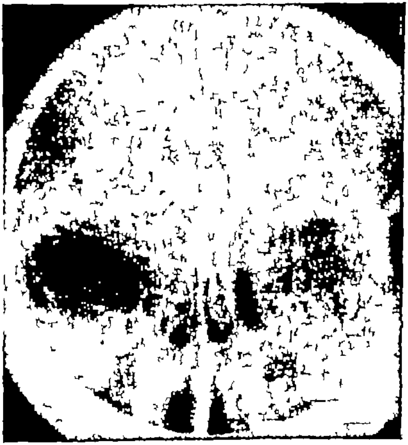

Fig 14 Case 4 M S. Preoperative X ray film showing small osteoma in a large sinus but with clinical symptoms.

In 1938, Malan collected 178 cases of osteoma of the frontal sinus.[13] In 1941, Leed[6] found 321 cases, a figure which he felt indicated that osteoma, while not common, is not rare.

Treatment These neoplasms are benign and without general serological or cytological changes. They occur and develop, as a rule, within a hollow chamber that accommodates them, usually for a matter of years, with no revealing symptoms. In due course, the slowly enlarging mass impinges on one or the other of the frontal sinus walls, so slowly, so gently, that resulting symptoms are not explosive or disruptive to the peace of mind of the victim. Obviously, with all the nearby vulnerable structures, definite symptomatology nearly always eventually occurs; in fact, the tendency is to malign encroachment on some of the neighborhood tissues[14] by erosion of the inner wall, the anterior wall, the orbital wall, or frequently, by interference with normal drainage.

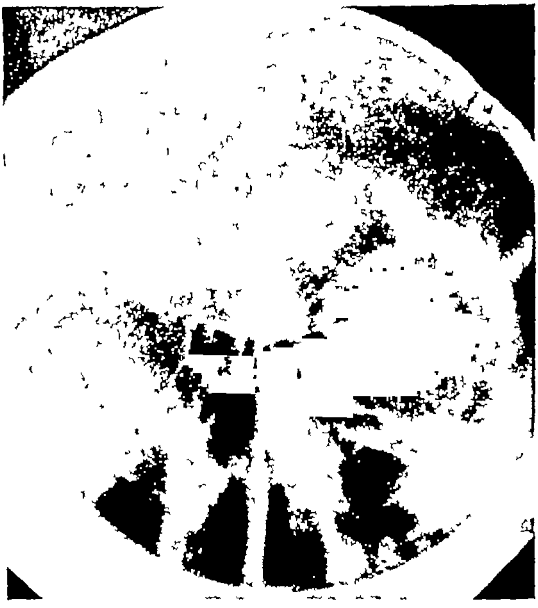

Fig 15 Case 4 M S Postoperative X-ray film showing osteoma gone and with the clinical symptoms relieved

Hart,[13] in 1943, reported a case that apparently sprang from the right ethmoid area and, before being studied by him, had extended to occupy a large part of both frontal sinuses.

In spite of this most extensive growth, the posterior walls of the frontal sinuses were (Roentgenologically) apparently intact; however, in the ensuing surgery one small area of bone erosion and dural exposure was found. Recovery was uneventful.

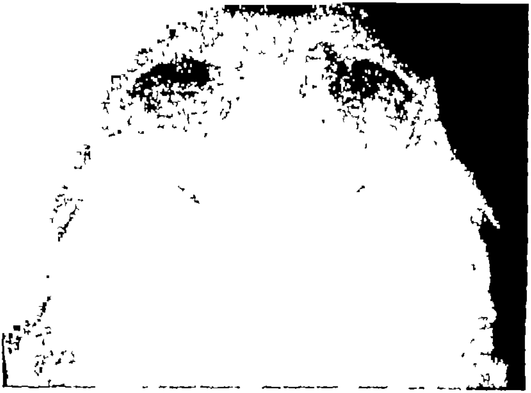

Fig 16 Case 5 E. H. X-ray film showing a very small osteoma in a very small sinus. The tumor is silent so far

Hart also quotes *Gatewood* and *Settel* as saying that up to 1898, only 20 cases had been attacked surgically, with a mortality of 45 per cent.

Begley and Hollberg[14] advise conservatism in considering *surgery. They agree that with definite signs and symptoms* present such treatment is positively indicated. Coates and Krauss[9] take the position that, being entirely surgical, as soon as there are enough symptoms to warrant a diagnosis the treatment should be immediate removal.

The chief symptoms are: external deformity; headache or pain, cerebral paralysis; nasal discharge; vertigo; proptosis; and diplopia. W. H. Johnston[16] reported a case in 1943, where the most prominent symptom was dizziness, the next being headache.

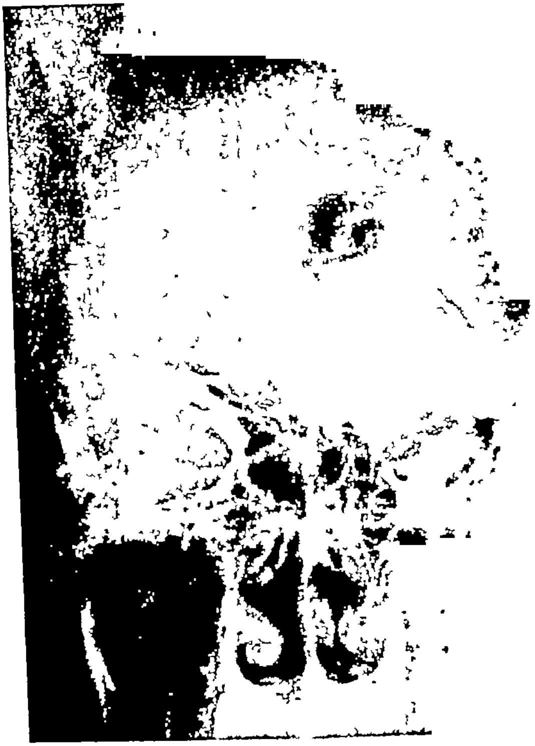

Fig. 17. Case 6. A. R. A-P X-ray film showing enormous bony tumor and peculiar shadows and apparent vacuum or cavity in the frontal cerebral lobe.

It is evident that usually when the tumor is relatively small in a capacious sinus, there is no justification for urging immediate surgery; however, the patient should be instructed as to the possibilities, and have an examination at least every two or three years. Case 3 establishes what may happen in the course of a dozen years. Case 5 shows that size of the tumor alone is not the criterion, as a very small tumor in a very small sinus may demand early interference. Certainly Cases 1 and 2 demonstrate signs and symptoms that positively necessitate no delay in surgical removal. Case 4 reveals a tumor evidently relatively small, with no apparent impingement, but with clinical symptoms which were relieved by surgery.

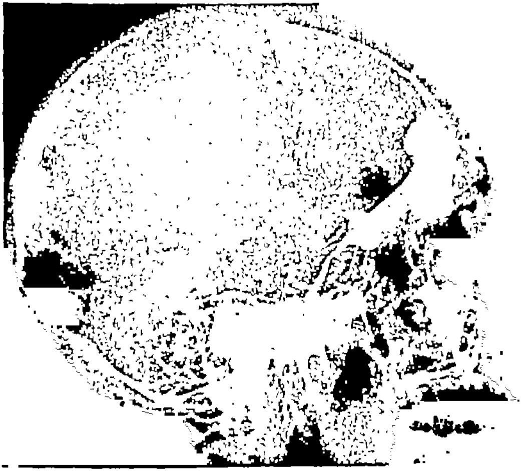

Fig. 18. Case 6. A. R. Lateral X-ray film showing tumor mass in frontal sinus, thickening or opacity in area of posterior plate, and peculiar abnormal shades and shadows in the frontal cerebral lobe.

Because of continuous, even though slow, growth, encroachment, erosion, pressure and encouragement of extension or invasion of infection, eventually bring on various symptoms, the possible extension and the resulting destruction it will bring is unpredictable. Secondary infection in unoperated cases has been the chief cause of high mortality in the past.[15]

Boher[17] points out that pneumoencephalus is likely to result whenever an air filled sinus, such as any of the paranasal sinuses, has an interruption in its continuity and air enters the subdural space or the subarachnoid space. In some cases the air may penetrate into the lumen of the ventricular system. He noted vomiting and papilledema as significant symptoms.

Case 6 makes clear the very serious complicating possibilities of neglect or overlooking of symptoms that should warn and indicate much earlier study and necessary surgical action.

Varying and doubtless in many cases quite long periods of silence are followed by clear-cut signs and symptoms. With the increase of X-ray study of sinuses, detection of these silent and unsuspected tumors has probably shown an equivalent increase. These studies have also increased the number of successful results in handling such growths.

In the years before antisepsis[10] the surgical mortality was 31.2 per cent, while since 1875 it has fallen to only 3.7 per cent.[5]

Surgical Procedures and Technique: In reviewing the literature of the past 20 or 30 years, when surgery is indicated, the various authors so state and proceed to remove surgically, without much discussion as to method or technique. It is probable that the usual surgery of the frontal sinus exemplified by the Killian, Lynch or Lothrop techniques was utilized, or the orbital approach mentioned by Benedict and Beck in a discussion in 1931.[8]

Pilcher,[19] in 1938, recorded a successful operation on an osteoma of the frontal sinus by the transcranial approach; however, in contrast to the general clinical reports, Vadala and Somers[20] made a thorough study of the surgical approach and technique.

1. A horseshoe temporal flap (Cushing) with a transcranial approach.
2. A curvilinear eyebrow incision with vertical extension (Conley). This was used largely to insert a plate to repair postoperative deformity.

3. An eyebrow butterfly incision with vertical extension (Hagoman).

4. The eyebrow butterfly incision bilaterally, without extension vertically (Hauley).

Any of these last three incisions should give adequate exposure, when indicated, either for osteoperiosteal flap or for insertion of a metal plate to repair postoperative defects.

5. One other is mentioned, a horizontal transfrontal incision (Durling), mainly of value if the tumor appears to be attached in the area of the frontal sinus septum

When the anterior sinus wall is removed, in case of a large tumor, the subsequent cosmetic result may be quite undesirable. In Case 1, the author used the typical Killian incision and removed the anterior plate of the right side, and to a moderate extent over to the left of the septum, so as fully to expose the tumor and to permit easier surgical attack. Even the right supraorbital ridge was so involved as to necessitate its sacrifice.

Twenty-two years later (Case 2) an attempt was made to prevent deformity by the *lid operation*, and in Case 3 a further improvement in technique was devised by using the flap or *hinge operation.* Illustrations of these three techniques, photographs and a moving picture film of Case 3 will be shown.

SIX CASES OF FRONTAL OSTEOMATA (EPITOMIZED).

Each showing a clear-cut difference in its pathological, clinical and surgical details.

Case 1 M. J Surgery was performed in August, 1928 at the Battle Creek (Mich.) Sanitarium with the assistance of Dr C G Wencke A large part of the anterior frontal plate and most of the right supraorbital ridge was removed, with resulting marked deformity

Case 2 E. J Operation performed on July 2, 1950 at the Glendale (Calif) Sanitarium and Hospital. This patient was referred to the author by Dr Leland R. House, who was unable to operate, due to an acute injury to his hand but was present as observer and adviser With the assistance of Dr Albert J Patt, the anterior frontal plate was removed, entirely and preserved as a lid to be replaced at the conclusion of the tumor removal.

Case 3: C. B. Surgery was performed on Dec. 4, 1950, at White Memorial Hospital. This patient was referred to the author by Dr. H. James Hara, who collaborated, and with the assistance of Dr. George Weyand, resident, a new technique was utilized, making a hinge of the frontal plate, base downward, and, thus, preserving a great part of the circulatory supply.

Case 4: M. S. This operation was performed by Dr. Leland R. House at the Los Angeles County General Hospital, June 9, 1949. It is used in this presentation by his consent and by permission of the General Hospital authorities, to illustrate a relatively small tumor, with clinical symptoms, relieved by surgery.

Case 5: E. H. This patient was referred to Dr. Leland R. House by Dr. A. L. Muff, for consultation and advice. By permission of Dr. House, the patient is presented in this series to illustrate a very small, and still silent, tumor in a very small frontal sinus, if indeed it really is frontal. The disproportion portends the necessity for relatively early removal of the tumor.

Case 6: A. R. This patient was operated upon by Dr. Wm. T. Grant, neurological surgeon, White Memorial Hospital, on Feb. 26, 1948. It illustrates certain possibilities of intracranial complications, in neglected cases, and the grave operative procedures thus necessitated.

None of these six cases has been previously reported.

PRESENTATION OF THE SIX CASES OF OSTEOMA OF THE FRONTAL SINUS IN DETAIL.

Case 1: August, 1928. Martin J., a sturdy sheet metal worker, age 30 years. His complaint was a bulging of the right eye, with constant diplopia. This had been observed several months, gradualy getting worse. There were no other symptoms. A tentative diagnosis of a neoplasm or mucocele behind the bulb was determined by X-ray study to be an osteoma of the right frontal sinus. This was confirmed by surgery. Incidentally, the mucocele was also found at this time. The procedure was performed under local anesthesia. The sphenopalatine area, the nasofrontal region and the middle meatus were packed with cocaine mud. A solution of novocaine with suprarenin was used to infiltrate the supraorbital region, blocking both the supraorbital nerves.

An incision was made beginning at the external end of the right eyebrow and through it to the nasal angle. This was extended down the side of the nose, across the bridge, up the left side of the nose and as far through the left eyebrow as the supraorbital notch.

An attempt was made to spare the anterior sinus plate, but this was not possible, as the tumor was found to completely fill the sinus, and, therefore, was lying immediately under the outer table. In the complete exposure of the tumor, which weighed 12.8 gm., it was necessary to remove all of the anterior wall of the right sinus and a portion of the anterior wall of the left. The septum between the two sinuses was practically destroyed by the pressure of the growth.

In order to remove the tumor, the right supraorbital ridge had to be sacrificed as certain tooth-like processes of the neoplasm had penetrated the floor of the sinus so close to the ridge that separation and conservation were not possible. The anterior ethmoid region contained another tooth-like projection about 1 cm. in diameter. The origin of the tumor was in the region of the right nasofrontal duct. Early in the surgical attack an attempt was made to bite off a portion of the upper edge of the tumor. A large mastoid rongeur was used, but under forcible closure of the handles the instrument broke, revealing the intense hardness of the bony mass. In exploring downward around the growth, an area was reached that was as surprisingly soft, having the consistency of maple sugar. A furrow was made in this soft bony tissue about the periphery of the hard growth. The osteoma was then easily rocked off and removed. This soft base was entirely cleaned away, down to hard, normal appearing bone.

In 1880, H. Knapp stated that one chisels around the base of the osteoma, "not attacking it from the surface or chiselling or sawing it off piecemeal."[11]

In 1892, Edward Jackson emphasized the "value of the drill when skillfully used, in effecting safe removal of part of bony growth where the base has to be left."[11] He refers, no doubt, to the sessile soft growths.

The right frontal duct was well opened. There was no mucosa evident in the right sinus. In the orbit, however, there were several polypi and a quantity of mucocele material. The mucosa of the left frontal sinus was also polypoid and was completely removed. The left nasofrontal duct was not touched. The remnant of the septum between the sinuses was removed down to the base so as to insure drainage of both sinuses through either side. Two small fenestrated rubber tubes were passed down *to the nasal vestibule through the right nasofrontal duct.* One tube extended upward and outward to the outer corner of the excavation made by the tumor. The upper end of the other tube lay free in the right frontal sinus. The tubes were gradually removed beginning on the third day. Surgical recovery and disappearance of the protrusion of the eye and of the diplopia ensued in a short time.

The patient valued the tumor so highly that he kept it in a bottle with alcohol. He returned to the clinic every few months to have the "alcohol changed." His wife recognized his evaluation of the tumor so well that when he died of pneumonia, in 1943, after 15 years, she placed the tumor in the casket with him; therefore, it cannot be demonstrated at this time.

The postoperative facial deformity was marked. Today it, no doubt, would be overcome by a tantalum implant; however, the defect did not seem to be particularly disturbing to the patient nor to his family.

Case 2: Mr. Elwyn J , age 76, was referred to Dr. Leland R. House by Dr. A. L. Muff, May 29, 1950. The patient had severe pain in the right frontal region and a somewhat lesser disturbance of the entire right half of the head. No evidence of sinusitis was found by a consultant at the time the pain began. The patient noticed a swelling over the right eye and that the skin in the same area was very sensitive, soft and yielding, not bony hard. In recent days he had developed a low grade fever. Penicillin seemed to afford some relief. Dr. Muff had an X-ray study of the sinuses made on May 28. This showed a density in the right frontal sinus which was diagnosed as a bony growth.

The patient first presented himself in person on July 2. Study of the accompanying film led to the confirmation of the diagnosis of a growth, ecifically an osteoma. The tender area of the skin was soft and evi- ently meant an area of erosion of the anterior sinus wall.

Appointment was made for surgery for the morning of July 24. The ening previous, Dr. House suffered an incapacitating injury to his ght hand. The author was asked to take charge and to conduct the eration. Dr. A. J. Patt and Dr. George Weyand acted as assistants. r. Colver, recalling the experience with Case 1, proposed to remove the terior sinus wall entirely as a cover or lid, preserve it in hot saline d replace it after the tumor was removed. There was a question hether the bone would "take," but it was felt that nothing was risked in aking the attempt. Dr. Albert J. Patt, following the measurements of a acing from the X-ray film, made an incision through the soft tissues d periosteum about the periphery of the sinus. Next, a series of small ill holes were made through the plate into the sinus. These holes were en connected by a smaller drill, making a perforating furrow. The terior frontal plate was then removed. Examination of the inner sur- ce showed an area of bone erosion, 12 mm. in diameter. The soft tissue this area was not normal and was curetted clean to the overlying ermal tissue. The thinned border of the circular eroded area in the one was then cut back by curette until the thickness and appearance of e new border indicated approximate normality. The plate was replaced a lid, and the periosteum was joined around the whole border with osely placed catgut sutures.

About a month later the whole area began to be painful, red, swollen d very tender. It was feared that the bone was breaking down, until fenestrated soft rubber tube was introduced from the lower border of e incision. Considerable pus was evacuated, but in the course of a few ays all inflammation subsided and the tube was removed. It was evi- ent that the area of traumatic and inflammatory erosion had had very w resistance and had succumbed to the resident bacteria. At no time as the sinus itself or the bone substance, in general, involved in the fection.

On Dec. 26 another X-ray study was made. (There had been two others nce the surgery.) The Roentgenologist reported: "There are still some idences of the bone fragments in the anterior portion of the right ontal sinus. There has, however, been considerable absorption of the one since the examination of Oct. 26, 1950. The burr holes are still ident in the upper portion of the frontal plate, above the absorbing ea of bone.

Conclusions: Continued absorption of the bone fragment forming the terior wall of the right frontal sinus. Whether or not the remaining one represents a sequestrum is difficult to say. It probably does, how- ver, and is gradually being absorbed.

The surgeon does not fully concur in this conclusion. Whether the one will absorb, be thrown off or survive sufficiently to make an effi- ent lid is still an open question. At present the whole area palpates ard, there being a dimple or retraction of skin over the area of erosion at was present before surgery.

Case 3: During the summer of 1950, Dr. H. James Hara learned of the rojected paper on osteoma. This recalled to him a patient that he had ad a number of years ago. At that time, in August, 1938, a Mrs. Caro- ne B. had consulted him in regard to nasal trouble. She was then 47 ears of age. She was found by X-ray study to have marked disease of e right maxillary sinus and of the right ethmoid labyrinth. A right

Caldwell Luc and a transantral ethmoidectomy were performed by Dr Hara, assisted by Dr Merrill O Dart, resident. During the course of the radiological study, there was observed a small osteoma of the left frontal sinus It measured about 1 cm or less in diameter and was silent.

In September, 1950, the patient, at this time aged 59, was located and was advised to have another X ray study This new film showed that the tumor had grown very considerably, nearly filling the sinus It appeared to be lobulated, the greatest diameter being 30 mm The patient desired to have this tumor removed

*After discussing the proposed surgery, involving a modification of tech*nique by Dr Colver, Dr Hara agreed to arrange for moving pictures of the operation. After preoperative medication intravenous sodium pentathol anesthesia was induced The eyelids of the left eye were closed by silk sutures. Novocaine infiltration was induced over the left eyebrow, across the bridge of the nose, and for a short distance along the right eyebrow A three-quarter butterfly incision was made and the skin elevated as thin as possible, leaving a thick subcutaneous layer of tissue over the periosteum of the frontal plate Bleeding was controlled by coagulation The uncovered area was demarked according to the size of the tumor, and the sinus outline, using the X ray film and a tracing as a guide About 12 penetrating burr holes were made by Dr George Weyand up the mesial border, across the superior border and down to the level of the brow along the outer border The line for the holes was exposed by an incision through the periosteum with minimum retraction. Instead of connecting the holes with drilling as was done in Case 2, they were joined by means of a sharp chisel, loosening, but conserving the bone substance Then the modification of technique was done by making two short incisions in the supraorbital line Through these incisions the chisel was insinuated and sharply tapped This broke or definitely weakened the bone of the plate horizontally, but spared the periosteum and connective tissue and thus the circulation from below It also insured the integrity of the supraorbital border Next, an elevator was passed along the line of bone incision on the lateral and upper borders By gentle loosening and then by prying or lifting, the horizontal line at the brow level was cracked and the frontal plate was swung forward and downward like a door, using the soft tissue as a hinge The tumor exposure and surgical accessibility were excellent. The tumor sprang from the ethmoid area. It was loosened by chisel and rather gentle tapping. This broke the stalk or base of the growth and it separated easily from its bed The soft base was adequately curetted. The tumor measured 33 mm. vertically and 43 mm. horizontally

The right frontal sinus was inspected through a tumor produced perforation of the septum. There was a moderate amount of greenish gray mucoid contents that was aspirated, emptying the right sinus There was no pus The mucosa apparently was normal The rest of the septum between the sinuses was removed No rubber tubes were passed and no dilating instrumentation was performed on the normally patulous frontal openings into the nose.

The hinged frontal plate was swung up into place. The lower border of overlying connective tissue seemed normal, the two short incisions not being readily visible A dozen catgut sutures were used to coapt the periosteal borders Silk sutures were used to close the skin incisions. A large pressure pad was applied over the left frontal area Postoperative progress has been uneventful. It is evident that the integrity of the bony cap has been insured through preservation of the circulation from below by the uncut thick connective tissue hinge

In the 1926 edition of the translated textbook of Prof. Hajek on Nasal Accessory Sinus, there is a section on radical operation by the osteoplastic method. The cases reported were purulent frontal sinusitis and not osteoma. Brieger, and later Shoenborn, were given credit for the first cases well over 50 years ago. The outcome of Brieger's case, unfortunately, was not given. He resected and reimplanted the osteoplastic flap. Shoenborn made a skin-bone flap with the base up. Scanty postoperative history was given. Czerny reported a case with cure of the suppurative sinusitis, effected in six months.

In 1897, Golovine reported three cases with base of the flap downward instead of upward. He considered one patient, at least, as cured after nine months. As to the cosmetic result, he said, "the traces of the operation remain scarcely visible."

Hajek states that "definite judgment regarding the value and duration of healing after the osteoplastic resection can be given only after years." The results in the hands of various surgeons were of varying success. The procedure seems to have fallen largely into disuse. As noted above, no case of osteoma, either with the base of the flap upward or downward, seems to have been handled by this method.

One essential difference between these early flaps and the one used in Case 3 is this: The early flaps were skin-bone. The skin incisions were made immediately to the bone and the whole thickness resected without too much regard as to cosmesis. In Case 3 the skin incisions were carefully made, the skin elevated upward and as thin as practical. The bone flaps were made, preserving as thick a layer of vascular bearing connective tissue as possible. The replacement suturing was in two layers, the bone and connective tissue and the skin.

There are two other cases, loaned to the author by Dr. Leland R. House, that illustrate the possible variations in relative size of the tumor and its containing sinus. They emphasize the necessity of careful clinical consideration in making a decision as to whether and when to advise surgery.

Case 4: Maria S., aet. 65, May 24, 1949. This patient was seen and later operated upon by Dr. Leland R. House, at Los Angeles County General Hospital. The case is presented to illustrate a tumor, relatively small, but demanding surgery to relieve annoying symptoms (local pain and left-sided headache). The X-ray film shows the smallness of the growth with a fairly large sinus.

Surgery was done on June 9, 1949, under local anesthesia. The Lynch type incision was made, the anterior plate opened and partially removed by chisel and drill. No special attempt was made to preserve this wall. The tumor measured 6 mm. in diameter. The base was just medial to the nasofrontal duct.

Surgical convalescence and recovery from the annoying clinical symptom was uneventful. A postoperative film was made and is also reproduced herewith.

Case 5: On Dec. 23, 1950, Dr. Leland R. House had a patient, Mr. Eric H., referred to him for consultation. No clinical details are available. The probability is that the case is symptomless as regards the frontal sinus. The right frontal sinus is so small that it has scarcely invaded the lower part of the frontal bone. Within it is a very small shadow, evidently an early osteoma.

The diameter of the growth is 5 or 6 mm. and the diameter of the sinus in that area is 8 or 9 mm. Here is a problem, indeed. It would seem that in spite of the smallness of the growth and its present silence, the relative absence of space for development would indicate the desirability of keeping early surgery in mind. It is probable that X-ray study at six-month intervals, together with the cooperation of the patient in observing the development of any clinical symptoms, will determine the therapeutic course to pursue. When the tumor impinges against the sinus walls or when disturbing symptoms develop, it is evident that surgical intervention will be clearly indicated.

Case 6: In February, 1948, Mr. A. R., age 30, was studied, by Dr. William T. Grant, neurologically and by X-ray film. A preoperative diagnosis was made of osteoma of the left frontal bone (sinus) and a glioma of the left frontal lobe.

On Feb. 26, surgical approach was made through the left temporofrontal area using a skin-bone osteoplastic flap, base out. The osteoma was removed in fragments. Its base was "in the anterior ethmoidal or in the medial part of the left frontal sinus." The osteoma was irregularly knobby. The posterior sinus plate was largely eroded, and the tumor was firmly attached to the dura "everywhere." The preoperative diagnosis of glioma was changed to a postoperative diagnosis of "cyst of the frontal lobe due to chronic irritation." This cyst was very large, containing a great quantity of gelatinous, yellowish fluid.

The surgical summary records: *Left frontal craniotomy with removal* of osteoma and amputation of left frontal lobe.

The history relates as a possible etiological factor and the resulting clinical course *1.* a fall with injury to the left forehead 17 years previous. *2.* Some sort of falling five years ago, beginning with severe headaches, nausea, occasional vomiting with drawing of the legs and with unconsciousness for a short while, occurring every two to three months until six weeks ago; and *3.* the past six weeks the attacks have occurred several times a day, the patient being unable to work.

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endoscopically. The gentle bronchoscopic aspiration of these patients frequently will prevent the necessity of tracheotomy and often is life-saving. It is also of great value to culture the aspirated material and test the organisms' sensitivity to antibiotics for best selection of an effective chemotherapeutic agent.

We, unfortunately, have had too little opportunity to treat asthmatics endoscopically to be able to make a critical evaluation.

Tubercular lesions are seen only diagnostically as their further treatment is carried out in sanitoriums.

The modern chest surgeon now offers much help and hope to a large class of patients who in the past have been largely neglected: those having esophageal lesions.

Congenital lesions are not too common; we have seen about three in the past two years. The congenital stenosis offers highest hope of cure; the fistula cases, as a rule, do not survive.

Diverticula is diagnosed by history, X-ray and esophagoscopy and are usually handled by surgical excision.

Foreign bodies like those of the bronchi are individual mechanical problems, and their variety is endless.

The lye burns are still seen too frequently. We follow a routine similar to that of Fisher and Hicks in the handling of early cases:

1. Wash stomach with mild acetic acid.
2. Tube feed if necessary.
3. Esophagoscope in four to five days to determine extent of lesion.
4. Do a gastrostomy and retrograde dilation of lesion, if the lesion is extensive.
5. Use Hurst's bougies if lesion is slight.

Most out of town cases are presented to us in a most unfortunate condition — emaciated, dehydrated and poor surgical risks. We treat them intravenously with blood or fluids, perform a gastrostomy, and start retrograde dilation as soon as practical.

Cardiospasm or achalasia is another obstructive lesion which may require the service of a chest surgeon. Most cases, however, respond to some form of dilation, either the hydrostatic or the Hurst bougie. We have several patients with their own bougies, using them as necessary.

Malignant disease of the esophagus is not too uncommon. All patients with persistent retrosternal discomfort and dysphagia should have contrast medium studies and esophagoscopy. Surgery offers these patients palliation and frequently cures.

I should like to say in closing my part of this presentation that I feel that the otolaryngologist doing endoscopy in the smaller cities has a responsibility which must not be shirked, in ever re-emphasizing to his fellow practitioners in his own and in dependent communities, particularly to the general practitioner, the need for the careful examination by every possible means of the "little cough," or the "little heart burn," and the "mild retrosternal pain." It is in that way only that we can hope to raise the statistical average of cures from malignant diseases of air and food passages.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

April 1, 1951.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver) ; Micronic Model 303. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp, 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 935 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Distributor: Sears-Roebuck Co., 925 S. Homan Ave, Chicago, 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 935 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Model 75; Zenith Miniature 75.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Meeting: Hotel Claridge, Atlantic City, N. J., May 9-10, 1951.

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Secretary: Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: Hotel Claridge, Atlantic City, N. J., May 6-8, 1951.

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Meeting: Hotel John Marshall, Richmond, Va., May 1-4, 1951.
Palmer House, Chicago, Ill., Oct. 9-12, 1951.

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Place: Army and Navy Club, Washington, D. C.

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Meeting: Chicago, Ill., Oct. 19, 1951.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros.

Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Havana, Cuba, January, 1952. For information write Dr. Chevalier L. Jackson, 3401 North Broad St., Philadelphia 40, Pa.

SECOND LATIN AMERICAN CONGRESS OF OTO-RHINO-LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

President: Prof. A. dePaula Santos.

Secretaries: Dr. Jose deRezende Barbosa and Dr. Plinio de Mattos Barretto, Hospital das Clinicas, Sao Paulo, Brazil.

Time and Place: Sao Paulo, Brazil, July, 1951.

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Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

Meeting: Hotel Claridge, Atlantic City, N. J., May 7-8, 1951, P. M.

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Secretary-Treasurer: Dr. Victor Goodhill.

Chairman of Section on Ophthalmology: Dr. Dennis V. Smith.

Secretary of Section on Ophthalmology: Dr. Carroll McCoy.

Chairman of Section on Otolaryngology: Dr. Howard P. House.

Secretary of Section on Otolaryngology: Dr. Edwin Scobee.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Blvd., Los Angeles, Calif.

Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

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President: Dr. G. M. Billings, Morganton, N. C.

Secretary and Treasurer: Dr. MacLean B. Leath, High Point, N. C.

Time and Place: Sept. 11-13, Hendersonville, N. C. Jointly with the South Carolina Society of Ophthalmology and Otolaryngology.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Augustus B. Dykman, 328 Medical Dental Bldg., Portland, Ore.

Secretary-Treasurer: Dr. Howard P. House, 1136 W. Sixth St., Los Angeles 17, Calif.

Meeting: Empress Hotel, Victoria, B. C., May 27-31, 1951.

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OSTEOMA OF THE PARANASAL SINUSES WITH REPORT OF THREE CASES.*

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Osteoma of the nasal cavity and paranasal sinuses is a well recognized clinical entity as witnessed by the extensive literature available on the subject; however, such literature as is available is concerned primarily with the gross and clinical aspects of the lesion and its surgical treatment. It is the author's plan, therefore, to emphasize the histopathology of this fairly uncommon tumor in his review of the literature and presentation of three cases.

INCIDENCE.

Although all bones of the body may be attacked, it is generally agreed that the bones of the paranasal sinuses are the most commonly involved. The statistics of Boenninghaus, completed by Dahman[1] (1922), enumerated 234 cases. Twenty years later, Malan[2] listed 458 cases of osteoma of the paranasal sinuses. Narrowed down still further, the frontal sinus is the most frequent site for the development of osteoma of the paranasal sinuses. Of the 458 cases reported by Malan, the frontal sinus was involved in 89 per cent, the ethmoid sinus in 24 per

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cent, the maxillary sinus in 9 per cent and the sphenoid sinus in 1 to 2 per cent. The statistics of Boenninghaus produce similar percentages. Teed[3] (1941) reported that a survey of the literature revealed 321 cases of osteoma of the frontal sinus alone. Unfortunately, there has been no comprehensive survey of the cases involving the ethmoid sinuses. From a review of the literature in 1938, Rawlins[4] reported only 27 cases of osteoma of the maxillary sinus. At that time he added two cases of his own and in 1940[5] reported an additional case, making a total of 30 cases. Osteoma of the sphenoid sinuses is extremely rare; in fact, Hallberg and Begley[6] reported in May, 1950, that they were unable to find a single case of proved osteoma originating in the sphenoid sinus.

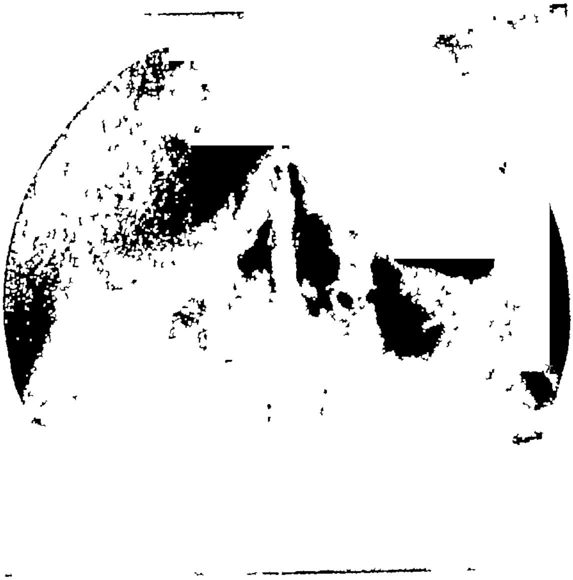

Fig 1 Case 1 Photograph of X-ray of sinuses showing the osteoma located in the outer three-fourths of the right frontal sinus

As to the incidence of the tumor in relation to sex, most authors have agreed that osteoma of the frontal sinus occurs more frequently in the male than in the female. In Teed's series on the frontal sinus, the proportion was 172 to 93. Nine cases of osteoma of the ethmoid sinus, of which six were male and three female, were included in the Hallberg-Begley series. According to Rawlins there has been no appreciable increased incidence in the male where the maxillary sinus has been involved.

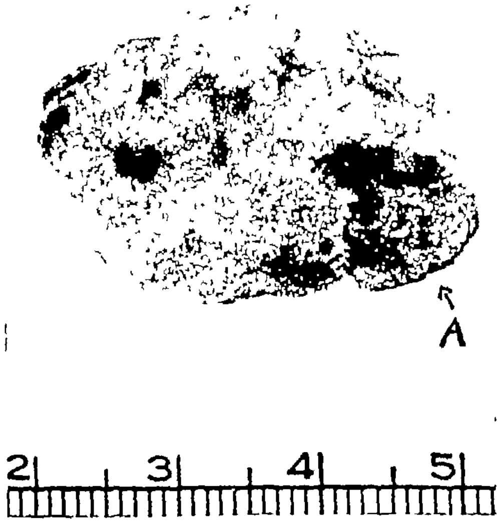

Fig. 2. Case 1. Photograph of osteoma. (A) Pedicle by which the tumor was attached to the anterior wall of the sinus at the lateral angle. It can be noted grossly that the pedicle is composed of spongy bone while the remaining surface of the tumor consists of dense bone.

Age distribution has been pointed out as one of the outstanding clinical features of osteomas. According to Armitage,[7] the tumor occurs in adolescence in 50 per cent of cases and before the age of 50 in 80 per cent. Among the cases reported and tabulated by Carmody,[8] the age range was from 11 to 61 years.

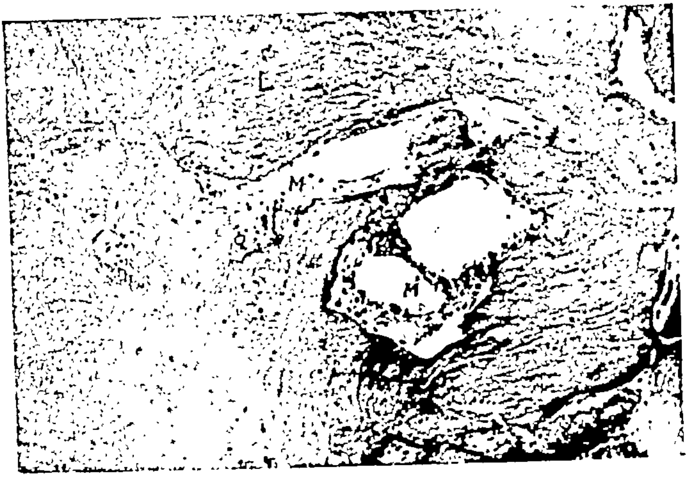

Fig. 3. Case 1. Photomicrograph (low power). (L) Lamellar bone; (M) marrow spaces filled with loose connective tissue and capillaries; (O) osteoblasts.

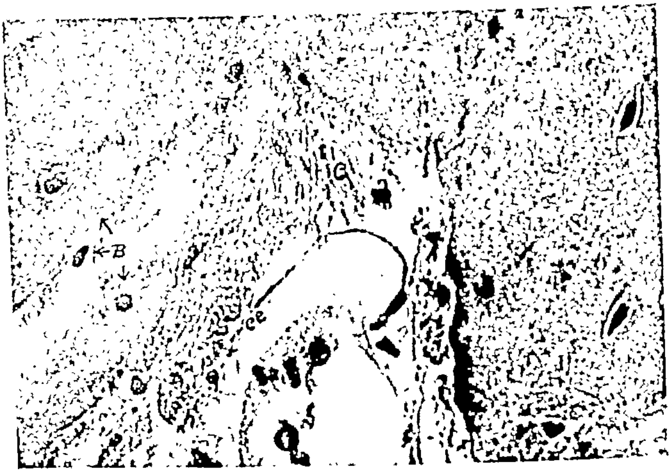

Fig. 4. Case 1. Photomicrograph (×400). (B) Bone corpuscles arranged without definite order; (C) loose connective tissue in marrow space; (cc) capillary within narrow space; (h) histiocyte.

ETIOLOGY.

Although many theories have been advanced, the exact etiology of this tumor is unknown. Some writers have stressed the importance of trauma; others have advanced the theory that repeated infections may be a factor. According to Fetissof,[9] the two most popular theories have been: *1.* development from embryonal cartilage and *2.* development from embryonal periosteum. Fetissof's work, which agrees with that of Pfeiffer (1912) and Eckert (1922), is the most convincing. From a histological study of four cases, he concluded:

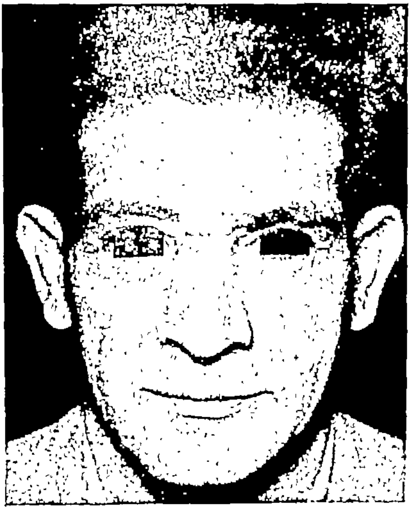

Fig. 5. Case 1. Photograph two months following operation.

1. The growth of the osteoma takes place from the inside outward by the metaplasia of fibrous tissue into bone tissue.

2. The osteoblastic ossificatory process that takes place at the periphery of the osteoma plays a secondary rôle in the growth of the tumor.

3. The fibrous tissue filling the interstices of spongiose bone is the direct continuation of the periosteum covering the osteoma from the outside.

4. The theory of origin of osteomas from the periosteum, split off at the stage of development of the organism of the periosteum, is the most fundamental.

Brunner and Spiesman[10] do not agree with Fetissof's theory that the spongiose portion of the osteoma is the more recently formed and that the compact bone represents an older part of the tumor. They state that in the expanding osteoma there is a slow but continual transformation of the compacta as well as the spongiosa and for that reason it is impossible to state which bone was formed first. They feel that the dense bone or solid cortex can also be deposited by the activity of the periosteum which forms the periosteal compacta. In other words, they apparently believe that the osteoblastic activity of the periosteum or submucosal periosteum at the periphery of the tumor plays a more important rôle in the bone formation

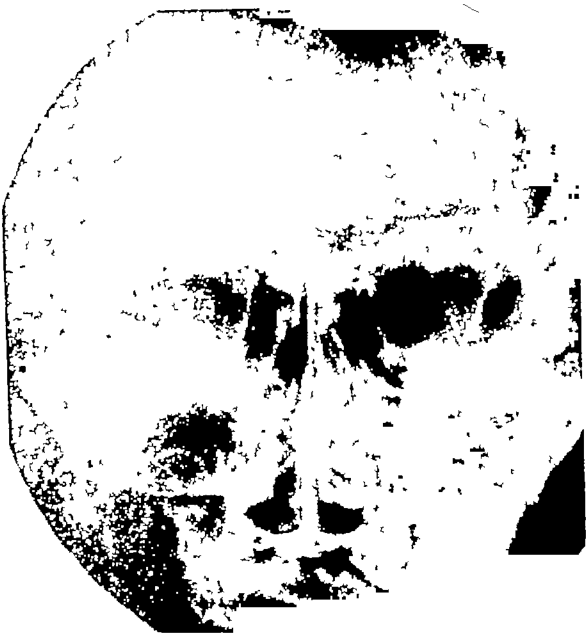

Fig 6 Case 2 Photograph of X-rays showing the osteoma located in the middle third of the right frontal sinus

or expansion of the tumor. Fetissof indicated that this took place but that it was of minor importance, that the main increase in size of the osteoma was through metaplasia of the connective tissue within the trabeculae of the spongiose portion which penetrated the tumor from the periosteum.

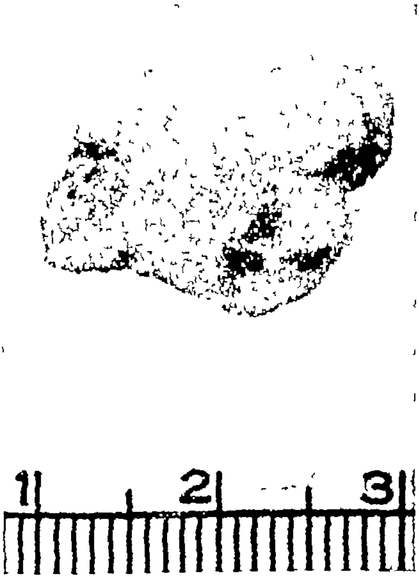

Fig 7 Case 2. Photograph of osteoma.

SYMPTOMS AND COMPLICATIONS.

The symptoms produced by osteoma of the paranasal sinuses depend upon the cavity involved and the size and extension of the tumor. The tumor is asymptomatic unless basal structures are pressed on or ventilation or drainage of the sinus involved is interfered with. In fact, the diagnosis

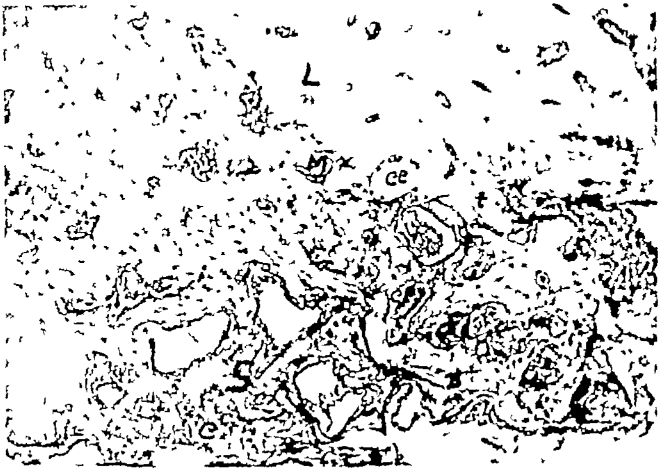

Fig 8 Case 2 Low power photomicrograph of osteoma (C) Connective tissue giving rise to osteoblastic ossification, (cc) capillaries within marrow spaces, (L) lamellar bone; (M) partially obliterated marrow spaces, (t) osteoid substance, (S) spongy bone with trabeculae

Fig 9 Case 2 Photograph three months following operation

is most commonly made as a result of taking routine X-rays of the sinuses. To illustrate, Childrey[11] has reported that 15 cases were noted in a review of 3,510 Roentgenograms of the sinuses, or an incidence of 0.43 per cent.

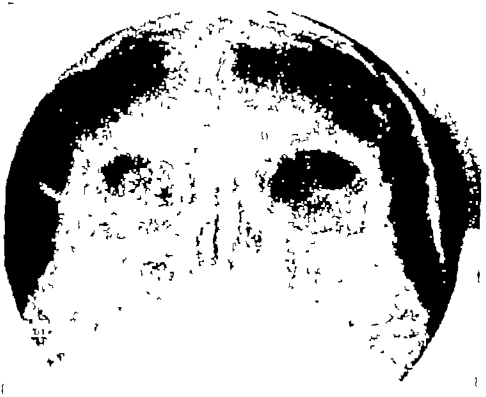

Fig. 10 Case 3 Photograph of X-rays of the sinuses showing the osteoma in the left infraorbital area

The classical symptoms of osteoma of the frontal sinus are dizziness, headache and deformity; however, ocular symptoms are not uncommon when the anterior wall and floor of the sinus are eroded. When such invasion of the orbit occurs, unilateral nonpulsating exophthalmos usually results with the globe depressed forward, downward and outward. Periorbital swelling, proptosis, diplopia and impaired vision are associated symptoms. Supraorbital or nasociliar neuralgia may also result.[12] If the direction of the growth is inward, the inner table is eroded, and the lesion becomes intracranial with resultant complications such as meningitis, brain abscess, or pneumocephalus. The latter may or may not be associated with cerebrospinal rhinorrhea. Growth into the region of the nasofrontal duct may isolate the sinus with the production of a mucocele or pyocele. In the series of 40 cases of osteoma of the frontal sinus prepared by Hallberg-Begley,[6] seven patients developed an infection of the frontal sinus on the involved side with resulting nasal discharge.

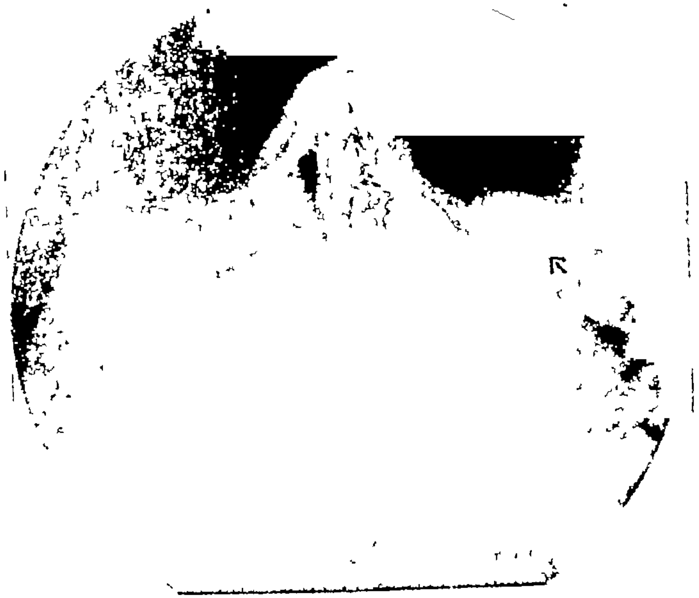

Fig 11 Case 3 Photograph of X-rays of the sinuses showing lipiodol in the left maxillary sinus and the osteoma arising from the left maxillary bone

Because of its restricted area, the ethmoid sinus produces symptoms earlier when attacked by osteoma than does the frontal sinus. Nasal obstruction and profuse nasal discharge on the involved side may be the earliest symptoms; however, the commonest symptom is frontal or maxillary pain. Again, orbital displacement is a symptom; but, in the case of ethmoidal osteoma, the globe usually is pushed forward and outward only, not downward as in the case of frontal involvement. Infection of the sinus is a complication. Cushing[13] has

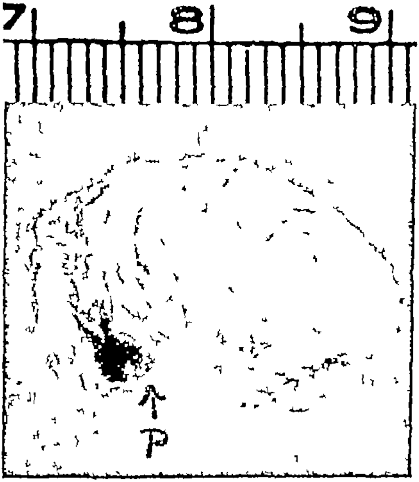

Fig 12 Case 3 Photograph of gross specimen (P) Pedicle

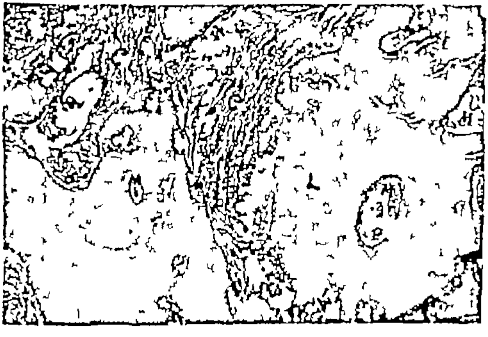

Fig. 13 Case 3 Low power photomicrograph of the osteoma (C) Connective tissue some of which is giving rise to bone by metaplasia (H) Howship's lacuna (L) lamellar bone containing bone corpuscles arranged without definite form (M) marrow spaces containing connective tissue capillaries and osteoblasts (O) osteoblasts.

pointed out that perhaps the majority of ethmoidal osteomata ultimately lead to intracranial complications of one sort or another. He reported three such cases, in one of which cerebrospinal rhinorrea was present due to communication with the cerebral ventricle. In a second case, a large intracerebral pneumatocele was demonstrated; and a large intradural mucocele was noted in the third case.

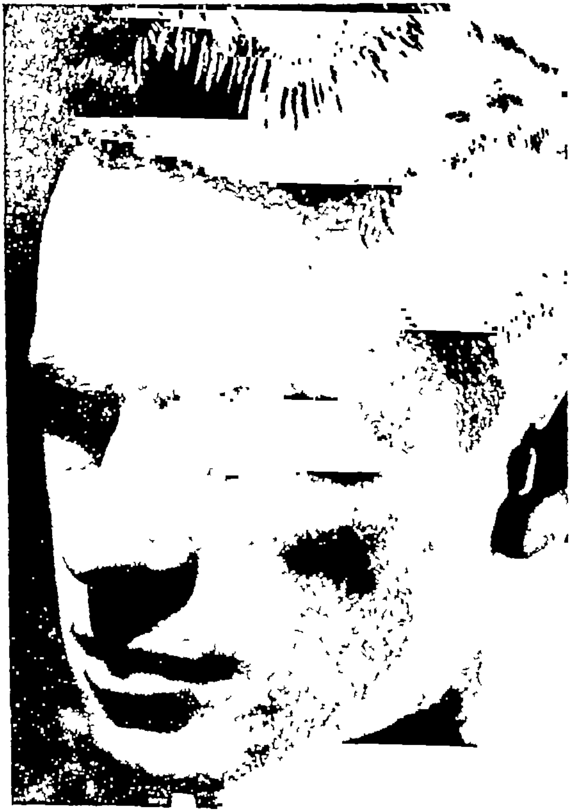

Fig 14 Case 3 Photograph showing a prominence in the left infraorbital area caused by the osteoma.

As in all cases of osteoma of the paranasal sinuses, the symptoms produced by maxillary involvement depend on the direction and extent of the pressure exerted by the tumor.

When it presses on the infraorbital nerve, neuralgic pain results. Pressure causing obstruction to the tear duct produces epiphora. Astigmatism and diplopia may be present from pressure on the eyeball; and pressure on the optic nerve may bring about impaired vision. Symptoms of deformity may take the form of swelling of the cheek, deflection of the nasal wall toward the septum, downward displacement of the hard palate, and upward, outward displacement of the eyeball. Exophthalmos often occurs. Suppuration of the antrum is common; mucocele is rare.

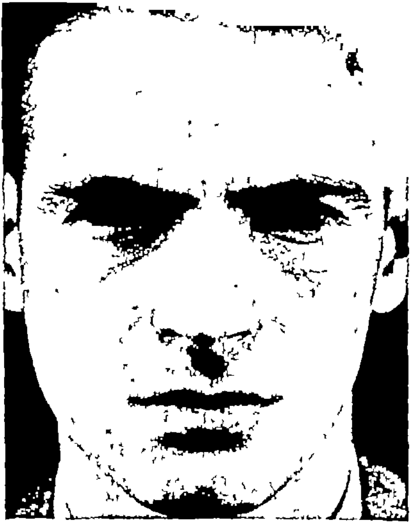

Fig 15 Case 2. Photograph taken three months after operation.

HISTOPATHOLOGY.

Osteoma is the term applied to tumors composed of bone and, as Ewing[14] pointed out, should be limited to true neoplasms. These tumors vary in size from a few grams to 440 grams, the latter occurring in a case reported by Hilton[15] in 1836.

The bones of the face, in addition to giving rise to true osteomas, also are the seat of a variety of bony overgrowths which may be difficult to classify. Osteomas should be differentiated from exostoses or circumscribed masses projecting above the bony surface and from exostoses which lie within compact or cancellous bone. From the standpoint of histopathology, one is unable, as a rule, to make such a differentiation; therefore, it is important to identify each on the basis of gross and clinical features since the osteoma represents a neoplasm and the capacity to destroy adjacent structures by expansion. Exostoses usually arise from an overgrowth of subperiosteal bone on the basis of some inflammatory change in the periosteum and represent hyperplastic bony phenomena. Some of the hyperplasias of the maxilla may be very easily confused with true osteomas of the maxilla or with those arising within the maxillary sinus.

Microscopically, three types of osteomas are described: *1.* osteomas made up of eburnated bone (osteoma eburneum), *2.* spongiose osteoma (osteoma spongiosum), and *3.* mixed osteomas (osteoma mixtum). The mixed-type osteomas are the most common; and, in fact, in most osteomas the periphery is made up of compact bone and the center is composed of spongiose.

Osteomas originating within the sinus cavities are covered with pseudostratified columnar ciliated epithelium beneath which is found the submucosa and periosteum. Brunner and Spiesman[15] stated that in their cases the microscopic findings indicated that the periosteal compacta or lamellar bone at the surface of the tumor was laid down by the paranasal mucosa which serves as a periosteum.

The microscopic picture of osteomas in general differs from that of normal bone in that in the compact areas dense lamellated bone with few or no Haversian canals is seen. The lamellae are arranged in layers parallel to the surface. The bony corpuscles are arranged without definite order. The *majority of the vascular canals run parallel to the long axis* of the tumor. In the spongy portions there are irregular groups of interlacing trabeculae surrounded by connective tissue growing into the bony structure. The connective tissue within the trabeculae contains a great number of fibres between which fibroblasts may be seen. As indicated by the work of Fetissof,[9] it is by metaplasia from such connective tissue that the formation of new portions of bone occurs.

The osteoma's pedicle is usually composed of spongy bone and is penetrated by the periosteum which may run the entire thickness of the tumor and pass into the connective tissue filling the interstices of the trabeculae.

TREATMENT.

Surgery is, undoubtedly, the only treatment for osteoma of the paranasal sinuses; however, as late as 1918, Mackenty (quoted by Novick[10]) considered an osteoma inoperable. Today, with our improved surgical technique and antimicrobial therapy, the tumors should no longer be left alone until they have invaded or displaced neighboring structures or caused complications which may endanger life. Joseph Beck[11] stated that 48 per cent of unoperated cases of osteoma would die of complications arising from the presence of the tumor. Although this figure has been labeled "unsupported" by some, the fact that surgery is the only treatment has not been altered.

There has been some divergence of opinion as to when surgical intervention should be undertaken. It is the author's *belief that once an osteoma has been demonstrated by X-ray* and there is evidence of active growth clinically or by X-ray examination, the tumor should be removed. If the lesion is small and its location is such that moderate growth does not threaten adjacent structures, then the growth of the lesion may be followed by X-ray examination at six month to one-year intervals. The case of a patient, age 15, reported by Coates and Krauss[18] demonstrates the rapidity with which these tumors can increase in size, particularly in the younger age group. Where possible, osteomas of the frontal sinus should be removed before the nasofrontal duct has been involved. To avoid intracranial complications, osteomas of the ethmoid sinus probably should be removed much earlier than those involving the frontal or maxillary sinuses.

In dealing with osteomas, one should remove not only the tumor but also its pedicle in order to avoid recurrence. Where the pedicle is attached to the posterior wall of the frontal sinus, it may be necessary to remove the posterior plate of the sinus; even so, there is little real danger and the plate will regenerate as it did in one of the cases reported by Coates and Krauss.[18]

Exposure of the osteoma may be accomplished by various incisions and approaches. For most of the frontal osteomas and for small lesions of the ethmoid sinus, various modifications of the Killian incision are most suitable. In the case of large osteomas of the orbitoethmoidal type with intracranial extension, with or without complications, it may be necessary to employ the intracranial approach as advocated by Cushing[13] or a combination of trans-sinal and intracranial approach. Osteomas of the maxillary sinus may be approached by either the Caldwell-Luc or the Denker methods.

CASE REPORTS.

Case 1: L. R., a white male, aged 30, was admitted to the otolaryngological service of Halloran Veterans Hospital the latter part of March, 1948. The patient complained of severe right frontal and right temporal headache. The headaches were first noticed in 1942 when they would occur at three to five-week intervals and last for two to three days. They were less marked in 1944 and 1945. From December, 1946, to January, 1948, the headaches were moderately severe, recurred every three to six weeks and lasted one and one-half to three days. The patient stated that they were continuous from January until March, 1948. They would be quite severe for eight to nine days, would ease off for two or three days and then become severe again. The patient could neither sleep nor do a full day's work during the severe attacks.

Routine X-ray of the paranasal sinuses revealed a large osteoma of the right frontal sinus which filled the lateral three-fourths of the sinus.

On April 1, 1948, the patient was operated upon under local anesthesia. The anterior wall and floor of the right frontal sinus were exposed by an incision along the lower margin of the eyebrow. The floor of the frontal sinus was entered at its inner angle, using the perforating burr. The tumor completely filled the outer three-fourths of the sinus and could not be removed *in toto* without sacrificing most of the anterior wall and floor of the frontal sinus. The growth had almost completely eroded the anterior wall and floor of the right frontal sinus in the area of the supraorbital foramen. The osteoma was attached by a small friable pedicle to *the anterior wall* at the *lateral angle of the sinus*. The pedicle and adjacent bone were removed. The posterior wall of the sinus was intact and covered by normal mucous membrane. The frontal ostium was not involved and was left untouched. There was normal mucous membrane about the frontal ostium for at least 1 cm. A small catheter drain was inserted at the outer angle and the wound closed in the routine manner. A pressure dressing was applied.

Twenty-four hours later the sinus was irrigated with penicillin solution colored with methylene blue. The solution freely entered the nose and the drain was removed. The wound healed per primam. Postoperatively the only subjective complaint was slight diplopia which soon disappeared.

The pathology report by Dr. Oscar Auerbach follows:

Macroscopic: Present is specimen of bone measuring 3 by 2 by 1.5 cm. The surfaces are lobulated and the lobules vary from 2 mm. to 7 mm. The surface is pinkish white and smooth for the most part, but one surface is rough. On section, the tissue is stony hard. The cut surface is white to pinkish white and the surface is rough.

Microscopic: Microscopic examination shows thick and irregular bony trabeculae with narrow marrow spaces. Within the marrow spaces are fibroblasts, histiocytes and some capillaries.

Diagnosis: Osteoma.

Case 3: E. P. C., a white male, aged 38, was admitted to the otolaryngological service of Halloran Veterans Hospital in May, 1948, with the chief complaint of right-sided headaches.

The patient gave a history of generalized headaches occurring from the age of 10 until the age of 17, at which time they disappeared. He again developed headaches in 1943 at the age of 29. At this time they were localized about the right eye with a tendency to radiate back to the right occipital area. At times there was a throbbing sensation in the right temporal area and about the right eye which would keep the patient from going to sleep. The headaches increased in severity until he was unable to work.

Routine X-rays of the sinuses revealed a large osteoma of the right frontal sinus measuring 12 by 15 mm. in the anterior-posterior projection and 1 cm. in the lateral projection.

On May 6, 1948, the patient was operated upon under local anesthesia. The incision was made along the lower margin of the right eyebrow. The floor of the frontal sinus was entered at its angle, using the perforating burr. The anterior wall of the frontal sinus was removed around the osteoma, using a small Kerrison rongeur. The tumor filled about one-half of the frontal sinus but did not encroach upon the frontal ostium. It was attached to the middle third of the posterior wall of the sinus by a very small pedicle. No destruction of adjacent bone was noted. The osteoma was easily freed and the base of the pedicle, which measured only about 3 to 4 mm. across, was destroyed with a polishing burr. The mucous membrane about the frontal ostium was normal and it was left intact as far out as possible, which was over 1 cm. A small catheter drain was inserted at the outer angle of the wound and the wound closed in the routine manner. A pressure dressing was applied.

Postoperatively the sinus was irrigated daily through the catheter drain with penicillin solution. The solution entered the nose freely on the third postoperative day and the drain was removed the following day. The patient developed a stitch abscess at the inner angle of the wound, which was easily drained and which did not involve the sinus. The convalescence was otherwise uneventful. Although the patient still experiences an occasional headache, he states that they are not of the same type or severity.

The pathology report by Dr. Oscar Auerbach follows:

Macroscopic: Present is a portion of lobulated bone measuring 2 by 1.5 by 1 cm. The surface for the most part is yellow-gray in color and smooth and in one area, 0.5 cm. in diameter, the surface is red-gray and roughened.

Microscopic: Microscopic examination shows thick and irregular calcified bony trabeculae with, for the most part, narrow marrow spaces. The spaces contain pink, dense connective tissue and the usual marrow cells are absent.

Diagnosis: Osteoma of frontal sinus.

Case 3: H. L., a white male, aged 24, was admitted to the otolaryngological service of Halloran Veterans Hospital, Dec. 15, 1949. The patient had complained of a "stinging feeling" in his left cheek for two months prior to admission. He had noticed a hard lump under his left eye which had increased in size during the past two and one-half years.

Routine X-rays of the sinuses and X-rays of the sinuses with lipiodal instilled into the left maxillary sinus showed a bony tumor over the left maxillary bone at the junction of the maxillary and zygomatic bones. The tumor did not appear to enter the maxillary sinus.

The local examination of the ears, nose and throat and the general physical examination were negative.

On Dec. 21, 1949, the patient was operated upon under local anesthesia. The osteoma was exposed through a small horizontal skin incision 1 cm. below the left infraorbital rim. The incision was carried through the soft tissues and the tumor was exposed lying in a shallow trough on the outer upper aspect of the maxillary bone. The osteoma was easily removed by introducing a curved gouge along the cleavage line found between the osteoma and its bed in the maxillary bone. The osteoma was attached by a definite pedicle at the suture line between the maxillary and zygomatic bones. The base of the pedicle was destroyed with the electric polishing burr. The skin was closed with a single subcuticular dermal suture.

The pathology report by Dr Oscar Auerbach follows

Macroscopic Present are two portions of bony tissue, the largest of which measures 2.1 by 1 4 by 1 cm. and is reniform in shape The smaller portion of bony tissue measures 1 by 0 5 by 0 5 cm. and is rounded in one aspect and irregular on the opposite aspect. The surface of the larger portion is composed of dense pink white bone which in some aspects is smooth while in other aspects it is slightly roughened.

Microscopic Microscopic examination shows a portion of bony tissue composed of wide dense trabeculae. These trabeculae vary greatly in thickness from segment to segment. In areas the mosaic lines are prominent. Some of the marrow spaces contain dense bands of connective tissue consisting of fibroblasts and capillaries while others are filled with fat.

Diagnosis Osteoma left infraorbital region.

COMMENTS

Three cases of osteomas of the facial bones are reported. All three cases occurred in males between the ages of 24 and 38 years In the first two cases the osteomas definitely arose from within the right frontal sinus, the pedicle arising from the anterior wall in the first case and from the posterior plate in the second In the third case the pedicle was attached to the outer surface of the left maxilla in the infraorbital area near the suture line between the maxillary and zygomatic bones. The tumor did not enter the cavity of the maxillary sinus and must be classified as an osteoma of the maxilla rather than of the maxillary sinus

There was evidence of active growth and expansion of the osteoma in at least two of the cases In the first case the tumor had almost eroded through the anterior wall and floor of the right frontal sinus and was giving rise to frontal headache which was presumed to be supraorbital nerve neuralgia and secondary to pressure from the tumor Case 3 revealed clinical evidence of appreciable growth of the osteoma during the two and one-half years prior to operation Although the tumor had attained considerable growth in the second case, we had no definite clinical or X-ray evidence to indicate the rapidity of growth, but due to the proximity of the tumor to the nasofrontal duct, it was deemed wise to remove the growth before further encroachment upon this area occurred.

In the two cases involving the right frontal sinus the nasofrontal duct had not been involved and the mucous membrane of the sinuses in this area was left intact and the nasofrontal duct was not disturbed. This allowed the mucous membrane to regenerate in a healthy functioning sinus. It is obvious that both cases were ideal for this form of treatment. Where osteomas of the frontal sinus are not diagnosed until they have involved the nasofrontal duct or, while under observation, are allowed to encroach on this area by growth, the incidence of complications is greatly increased. Even if the tumor is completely removed by the Lynch-type frontoethmoid operation where the nasofrontal duct is necessarily altered, the chances of a mucocele or pyocele developing within a period of 10 to 12 years are quite good.

The removal of the osteoma in the third case presented no real problem and the cosmetic result was reasonably good.

Grossly and microscopically, the three osteomas reported here showed evidence of both dense, lamellar bone and spongy type bone, the pedicles consisting primarily of spongy bone and the peripheral portions of the more dense lamellar bone; hence, the three would be classified as mixed osteomas. A typical section showing both dense osteoid bone and spongy bone is shown in Fig. 8, Case 2.

Bone formation appeared to be derived primarily from metaplasia of the connective tissue penetrating the trabeculae from the pedicle (see Fig. 13, Case 3). Some bone formation probably occurred from the periosteum at the periphery of the tumor by an osteoblastic ossificatory process; however, no definite observations could be made on this point due to the limited material which was sectioned by the technician from each tumor.

CONCLUSIONS.

1. Osteoma of the paranasal sinuses is a fairly uncommon tumor. The frontal sinus is most frequently involved.

2. Fetissof's theory of origin of osteomas from the periosteum is the theory most acceptable.

3. Patients having symptoms suggesting osteoma of the paranasal sinuses should have routine X-rays of the sinuses.

4. Evidence of an increase in the size of an osteoma sufficient to threaten adjacent structures is an indication for surgical removal.

5. Surgery is the only treatment for these lesions, and care should be exercised to remove not only the tumor but also the pedicle to avoid recurrence.

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RECRUITMENT OF LOUDNESS IN THE DIFFERENTIAL DIAGNOSIS OF END-ORGAN AND NERVE FIBRE DEAFNESS.*

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In 1936, Fowler[3] described a method of testing hearing which was said to differentiate neural from conduction deafness without recourse to determinations of the thresholds of either bone conduction or air conduction of sound. This test consists of the measurement of the recruitment of loudness with increasing intensity of sound. Fowler first demonstrated the presence of recruitment by balancing the loudness of a sound which was alternately thrown first into one ear and then into the other. This was referred to as the "alternate binaural loudness balance" test.

Fowler noted varying differences in loudness necessary for balance in various types of deafness. He found that the relative sensation of loudness did not change in the two ears at any intensity of sound in conduction deafness. On the other hand, he observed in some cases of nerve deafness that the ear with the poorer hearing could hear pure tones more and more distinctly as the intensity was increased. In other words, loudness recruitment was present. "Nerve deafness" was used by Fowler in the broadest sense and included lesions of the organ of Corti as well as of the nerve fibres and ganglion cells.

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†Abstract of thesis submitted by Dr. Eby to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Otolaryngology and Rhinology.

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The phenomenon of the recovery of relative capacity to hear at higher levels of intensity by an ear which fails to hear at threshold of intensity is referred to as the loudness recruitment phenomenon[14]

When Dix, Hallpike and Hood,[5] in 1948, observed that the loudness recruitment phenomenon was characteristically present in Ménière's disease and absent in cases of VIIIth nerve tumors and lesions at the cerebellopontine angle, they gave new importance to recruitment tests in the differential diagnosis of lesions of the end-organ and nerve fibres which produce deafness

The same authors,[6] one year later, reported that further observations in two cases of Méniere's disease and two cases of neurofibromata of the VIIIth nerve revealed differences in the intelligibility of speech associated with the two lesions They found that, in Méniere's disease there was a recruitment of loudness but not recruitment of speech intelligibility, on the other hand, in the presence of neurofibromata, speech became more intelligible with increasing amplification up to a point These findings sharply contradicted previous concepts as to the physiologic mechanism of recruitment. For this reason confirmation of the hypothesis that recruitment of loudness is present in a lesion of the epithelium of the end-organ, whereas recruitment of loudness is not present in a lesion of the nerve fibres or ganglion cells of the VIIIth nerve, was considered necessary

In our study, alternate binaural loudness balance tests were performed on 32 patients with unilateral deafness diagnosed as Ménière's disease (end-organ lesion), on one patient with unilateral deafness thought to be the result of a vascular accident involving the organ of Corti, and on 16 patients with unilateral auditory nerve deafness due to a variety of causes.

In Table 1 is given the diagnosis in the 16 cases of nerve deafness in which alternate binaural loudness balance tests were made. The diagnosis of nerve fibre involvement was substantiated by the operative findings in eight cases VIIIth

TABLE 1.

LOUDNESS RECRUITMENT AND DIPLACUSIS BINAURALIS DYSHARMONICA IN 16 CASES OF NERVE FIBRE DEAFNESS.

Case	Diagnosis	Diplacusis	Loudness Recruitment
1	Neurofibroma of right acoustic nerve	—	—
2	Meningioma at right cerebellopontine angle	Present	—
3	Osteoma at right cerebellopontine angle	—	—
4	Neurofibroma of right acoustic nerve	—	—
5	Meningioma at left cerebellopontine angle	—	—
6	Astrocytoma of left temporoparietal lobe	—	—
7	Left intracerebral hematoma, secondary to arteriovenous fistula or angioma	—	—
8	Neurofibroma of right acoustic nerve	—	—
9	Paget's disease of the skull	—	—
10	Pneumococcic meningitis	—	—
11	Meningitis	—	—
12	Thrombosis of anterior inferior cerebellar or basilar artery	—	Incomplete
13	Probable brain tumor with resulting VIIIth nerve damage	—	—
14	Possible VIIIth nerve tumor	—	Incomplete
15	Vascular accident involving VIIIth nerve fibres	—	—
16	Parkinson's disease, cochlear nerve damage due to disturbed blood supply	—	Incomplete

nerve tumors and lesions in the cerebellopontine angle were found to be present. In the eight other cases in which the VIIIth nerve fibres were presumed to be affected but operation was not performed, the diagnosis was based on Roentgenologic, neurologic and otologic findings.

Speech intelligibility tests also were made on both groups of patients whenever such tests were possible.

EQUIPMENT AND TESTING METHODS.

The routine audiometric tests were made on an Audio-Development Co. Audiometer, Model 50E, by an experienced technician in a quiet room.

Alternate binaural loudness balance and speech intelligibility tests were carried out in a sound insulated room in which there was a noise level of approximately 35 db when tested with a general radio sound level meter These tests were made with a Maico E1 Audiometer Thresholds for speech reception were determined by use of the spondee word test developed by Hudgins and his co-workers Speech intelligibility was determined by use of the phonetically balanced (P B) word lists worked out by Egan and his group at the Psycho-Acoustic Laboratory of Harvard University

When speech intelligibility tests were made, the microphone was held 2 inches (5 08 cm) from the examiner's lips The intensity of the voice and inflection were maintained as constant as possible with the assistance of a speech monitoring meter (Maico Part No 4506) With the meter adjusted to the test conditions, the voice was controlled in order to cause a swing of the meter pointer to the 0 db point on the carrying phase preceding each word Then each word of the phonetically balanced (P B) word list was allowed to vary according to its own phonetic composition

The intensity level at which speech sounds became uncomfortable to the listener was taken as the threshold of discomfort for the ear

RESULTS

Loudness recruitment was absent on the alternate binaural loudness balance test in the eight cases of nerve deafness in which the diagnosis was made at operation and in all but three of the remaining eight cases of nerve lesions In the three cases in which some evidence of recruitment was found, it was of only a slight degree and in no instance was recruitment complete

In 15 of the 16 cases of nerve fibre deafness, diplacusis binauralis dysharmonica was absent.

In Fig 1 are shown an audiogram and the results of the alternate binaural loudness balance tests on a patient who had a neurofibroma of the VIIIth nerve. The absence of loudness recruitment is indicated by the almost parallel loudness balance lines joining the sound intensities which produced equal loudness in the two ears. Audiograms in other cases were similar (see Figs. 2 and 3).

The results of alternate binaural loudness balance tests on 33 patients whose deafness was presumed to be due to lesions of the end-organ of hearing are given in Table 2. Loudness

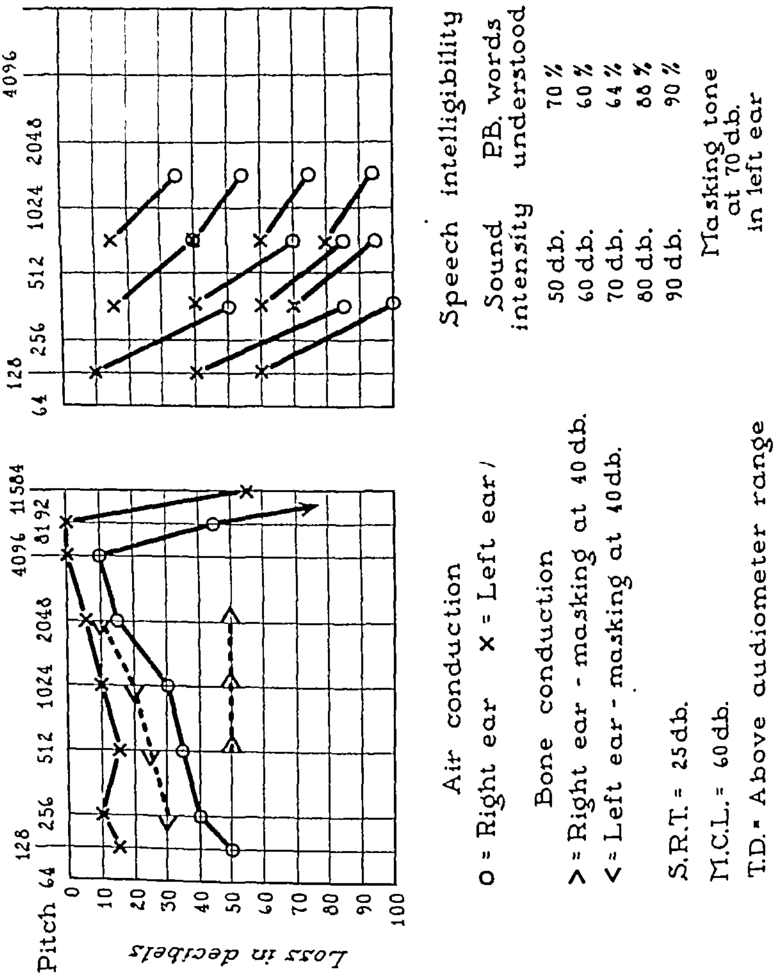
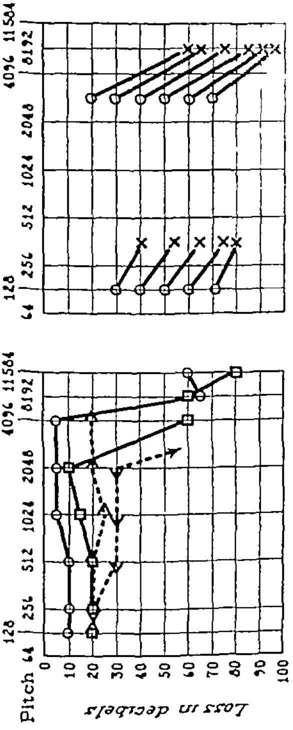


FIG. 1. Case 1, Table 1. Audiogram in a case of neurofibroma of the VIIIth nerve giving results of the alternate binaural loudness balance and speech intelligibility tests.

recruitment was present in every case. In only two cases was recruitment found to be incomplete within the limits of the audiometric scale. In 17 of the 33 cases, over-recruitment was expressed on the test. When a tone is heard by the defective ear with loudness equal to that heard in the normal ear even though the intensity of sound delivered to the defective ear is less, over-recruitment is said to be present.



Air conduction

O = Right ear X = Left ear

□ = Left ear - masking at 40 db

Bone conduction

> = Right ear

< = Left ear - masking at 40 db

SRT = 10 db

MCL = 40 db

TD = Above audiometer range

Speech intelligibility

Sound intensity	PB words understood
20 db	72%
40 db.	88%
60 db	90%
70 db	92%
80 db	92%

Fig. 2. Case 6, Table 1. Audiogram in a case of astrocytoma of the temporoparietal lobe producing pressure on the VIIIth nerve. Results of the alternate binaural loudness balance and speech intelligibility tests are shown.

Twenty-seven of the 33 patients were tested for diplacusis binauralis dysharmonica. It was present in 23. Diplacusis binauralis dysharmonica is usually associated with a disorder of the end-organ of hearing.

An audiogram and the results of the alternate binaural loudness balance tests in a case of Ménière's disease are shown in Fig. 2. The presence of loudness recruitment is shown by

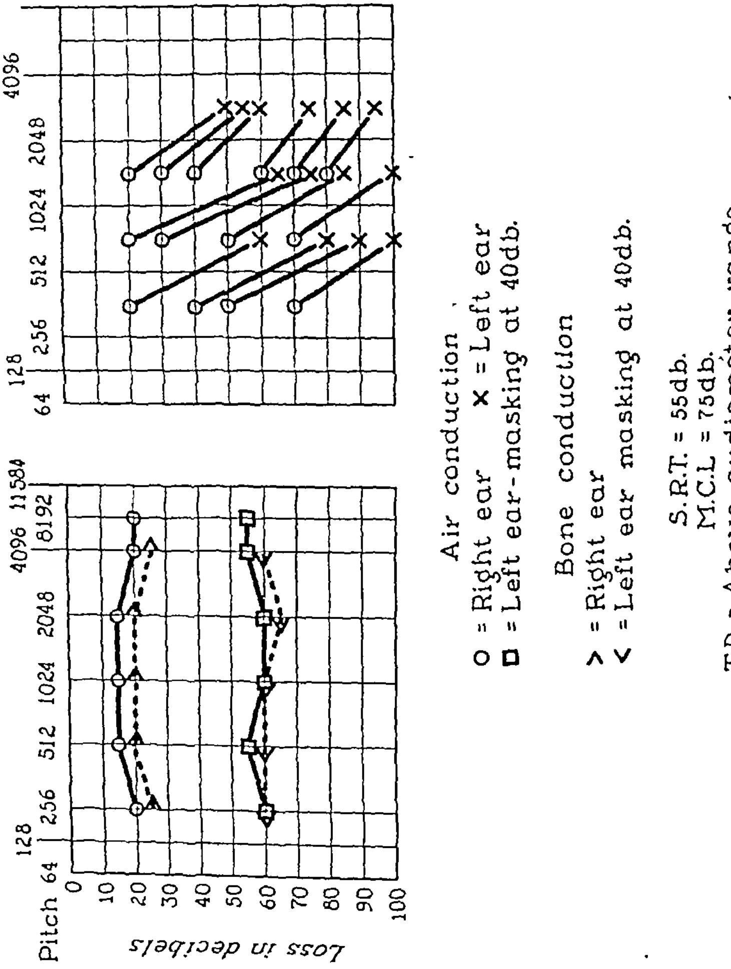


FIG. 3. Case 7, Table 1. Audiograms in a case of a hematoma in the left temporoparietal lobe secondary to an arteriovenous fistula or angioma. Results of the alternate binaural loudness and speech intelligibility tests are shown.

TABLE 2.

SUMMARY OF FINDINGS OF LOUDNESS RECRUITMENT AND DIPLACUSIS BINAURALIS DISHARMONICA IN CASES OF END-ORGAN DEAFNESS.

Cases		Total Cases
Loudness recruitment		33
Incomplete	2	
Complete	31	
Over-recruitment	17	
Diplacusis		27
Present	23	
Absent	4	

the loudness balance lines which become shorter and more horizontal with each increase of intensity until complete recruitment is reached.

Speech Tests.—In six cases of nerve fibre deafness speech intelligibility was tested by use of phonetically balanced (P. B.) word lists. In five of these cases speech became more and more intelligible as the intensity of sound was increased until the limit of the audiometric scale was reached. An example of such observations in a case of VIIIth nerve neurofibroma is shown in Fig. 1.

In 15 of the 17 cases in which a lesion of the end-organ of hearing was presumed to be present and these tests were made, speech intelligibility reached a maximum, then declined as the intensity of sound was increased further within the limits of intensity of the audiometer. An example of this observation in a representative case of Ménière's disease is shown (see Fig. 4).

The dynamic range for speech was narrowed in cases of end-organ deafness. In 19 cases the average difference between the threshold for speech reception and the most comfortable listening level was 18.42 db. This finding was in contrast to the wider span between these two levels found in seven cases of nerve fibre deafness in which the span was determined. In these cases the average difference was 28.57 db.

The threshold of discomfort for speech was determined in 12 cases of Ménière's disease and in one case in which deafness was thought to be due to a lesion in the organ of Corti. Of this group, the threshold for discomfort was found to be relatively lower than that found in the seven cases of nerve fibre deafness in which the test was made. In seven cases of nerve fibre deafness the threshold of discomfort was found to be beyond the limit of the audiometric scale.

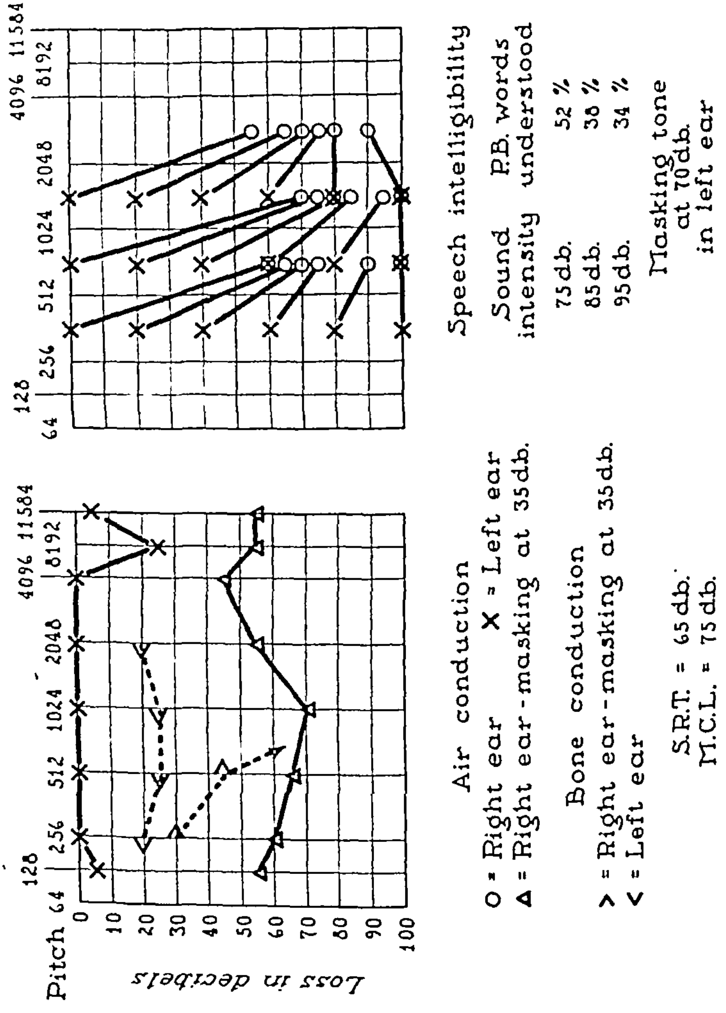


FIG. 4. Audiogram in a case of Ménière's disease with the results of alternate binaural loudness balance and speech intelligibility tests.

COMMENT.

In a discussion given on the recruitment of loudness phenomenon at San Francisco, in 1950, Fowler[6] differed sharply with the conclusion of Dix, Hallpike and Hood.[5] He stated that in performing loudness balance tests, if one ear has normal hearing and the other is deafened by a neural or so-called nerve deafness, the difference between the two ears at threshold will disappear as the sound is increased over threshold until at high intensities the sound appears just as loud, or sometimes even louder, to the deafened ear as it does to the normal ear. This pathologic increase in the increment of loudness is called "recruitment of loudness" and is pathognomonic of auditory neural lesions.

In regard to the report of Dix, Hallpike and Hood, Fowler stated that their hypothesis that recruitment arises only in lesions of the end-organ and, therefore, its absence in neural deafness provided a test for the differentiation between end-organ and neural degeneration deafness was interesting and he wished that it could be true. He, however, had never observed it unless there was insufficient hearing to permit the test. He offered as evidence an audiogram taken before operation in a case of neurofibroma of the VIIIth nerve in which recruitment was clearly shown.

Fowler also embraced the hypothesis of von Bekesy that stationary or increased difference limen arises from the same fundamental physiologic disorder as does recruitment of loudness. Our findings on testing patients with degenerative nerve lesions and presumed endolymphatic hydrops are diametrically opposed to the opinion expressed by Fowler and are in agreement with the findings of Dix, Hallpike and Hood.[4]

It is of great importance that this question be settled, because in the differential diagnosis of Ménière's disease, if one can have complete confidence in the differentiating value of the loudness balance test a forward step in the ease of differentiation of this disorder from conditions likely to be confused with it will have been taken.

In our experience at the Mayo Clinic we have not as yet been led astray by the loudness balance test. In one instance in which neurosurgical exploration of the cerebellopontine angle was done because of conflicting neurologic findings in the presence of loudness balance test showing complete recruitment no neural lesion was found and an intracranial division of the VIIIth cranial nerve relieved the symptoms.

Recent evidence presented tends to cast doubt on Fowler's assumption that an increased or unchanged difference limen and the recruitment of loudness phenomenon are different aspects of the same underlying physiologic change.

When the intensity of a sound is increased continuously, the corresponding sensation of loudness does not increase continuously but in discrete steps. A change in intensity sufficient to produce a just perceptible change of loudness is called "the intensity difference limen for loudness." It is well known that the normal intensity difference limen for loudness is relatively large at low intensities of sensation and much smaller at higher intensities of sensation. When recruitment is assumed to be present, the difference limen is much smaller than normal at low intensities and increases to normal values at higher intensities; therefore, if the difference limen increases or remains unchanged with increasing levels (intensities) of sensation, it is assumed that recruitment is present. If, on the other hand, the difference limen decreases with increasing levels of sensation, it is assumed that there is no recruitment.[4]

As found by Fowler,[9] von Bekesy and others, recruitment of loudness is common to both lesions of the epithelial end-organ and lesions of the nerve fibres and ganglion cells. Dix, Hallpike and Hood,[5] de Bruine-Altes, Mygind,[11,12] and now one of us (Eby), have found that recruitment of loudness is not present in nerve fibre and ganglion cell lesions. This indicates that there is some important difference in physiologic backgrounds of changes in difference limen and recruitment of loudness. It is for this reason that we believe Fowler's assumption to be incorrect.

Fowler's[9] theoretical explanation fits the conditions of change in difference limen perfectly, although as he pointed out it is unsatisfactory for explanation of the phenomenon of hyper-recruitment or over-recruitment.

Probably the hypothesis suggested by Bustamante Gurria and Garibay best explains the difference in sudden and slower changes in difference limen. Von Bekesy concluded from his investigation that reduction in the number of functioning nerve cells in a given area of the receptor organ of hearing would be indicated by an increasing of the difference limen. Mygind[11] considered von Bekesy's explanation completely acceptable for neural lesions. Mygind[11] accepted Shambaugh's suggestion that the tectorial membrane is the principal factor in analysis of tone. He pointed out that the nerve apparatus has been repeatedly found completely normal in disorders of hearing due to changes in the tectorial membrane; however, in changes in the tectorial membrane the distribution of the secondary sound waves on the organ of Corti show an unregulated pattern of discontinuous waves producing the same effect on the sensation of loudness as is produced by irregular degeneration of nerve fibres or ganglion cells.

Whatever the virtue, or lack of it, in Mygind's[11] theoretic explanation of the reason, change of difference limen can be found in both nerve deafness and end-organ deafness, the evidence submitted seems to be convincing that while diplacusis and recruitment are different aspects of the same underlying process, a stationary or decreased difference limen results from some different physical derangement.

We believe, therefore, that speech intelligibility tests, like the loudness balance tests, may be considered helpful in distinguishing end-organ from nerve fibre deafness.

SUMMARY AND CONCLUSIONS.

Alternate binaural loudness balance tests were made on 32 patients with unilateral deafness diagnosed as Ménière's disease, and on one patient with unilateral deafness thought to be the result of a vascular accident which involved the organ of Corti. Loudness recruitment was present in every case. In 17 cases there was evidence of over-recruitment.

Alternate binaural loudness balance tests also were made on 16 patients with deafness due to degeneration of nerve fibres. In eight cases intracranial lesions which involved the VIIIth nerve were found at operation. In all eight, loudness recruitment was found to be absent. In eight other cases in which there was a probable lesion of VIIIth nerve fibres, there was little or no evidence of loudness recruitment.

These findings add further evidence to indicate that loudness recruitment is present in a lesion of the organ of Corti but absent in a lesion of the auditory nerve fibres.

Diplacusis binauralis dysharmonica was present in 23 of 27 cases of end-organ deafness in which this was tested, whereas it was present in only one out of 16 cases of nerve fibre deafness.

Speech intelligibility tests were made in 17 cases of deafness in which it can be presumed that a lesion of the end-organ was present. In 15 of these 17 cases, speech intelligibility reached a maximum, then declined with further increases of intensity within the intensity range of the audiometer.

Speech intelligibility tests were made in six cases of nerve fibre degeneration. In five of these cases, speech continued to become more and more intelligible with increased intensity of sound, until the limit of the audiometric scale was reached.

It would seem from these findings that discrimination deafness is an expression of the same fundamental disorder which is responsible for recruitment.

In 19 cases of end-organ deafness in which the speech reception threshold and most comfortable listening level were determined, the average difference in intensity of sound between these two levels was 18.42 db. In the seven cases of nerve fibre deafness in which these levels were determined, there was a wider span between them, an average difference of 28 57 db.

The threshold of discomfort for speech was determined in 12 cases of deafness due to Menière's disease and in one case of deafness due to a vascular accident involving the organ of Corti. In every case this threshold was found to be within the limits of the audiometric scale In the seven cases of nerve fibre deafness in which an attempt was made to determine this threshold, it was found in each instance to be beyond the limit of the audiometric scale

These findings, the early decline in the speech intelligibility, the decrease in the span of speech intelligibility, and the lower threshold of discomfort for speech in our cases of end-organ deafness seem to indicate the presence of loudness recruitment. In contrast, the results of speech tests in our series of cases of nerve fibre deafness seem to indicate the absence of loudness recruitment.

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OBSERVATIONS ON THE LOCAL USE OF CORTISONE IN THE NOSE IN ALLERGIC RHINITIS.*

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As otolaryngologists we have been intrigued by the multiplicity of clinical conditions which have been improved by the parenteral use of cortisone and ACTH. Reports in the literature on allergic conditions, such as asthma, being improved, and polypi in the nose and sinuses disappearing after administration of ACTH and cortisone made us feel that one or both of these might prove of benefit in allergic rhinitis. Bordley[1] found that ACTH given parenterally has a decided effect on nasal mucosa and states "in those patients with pale edematous polypoid nasal membranes the edema rapidly disappeared and the color became bluish-pink." The exact mode of action of cortisone on the tissues is not definitely known, but it is believed that, as a result of a blocking or neutralizing effect on the antigen-antibody reaction, there might be a local tissue desensitization.

The division of ophthalmology of our hospital discovered that topical cortisone used locally in the eye was as successful as parenteral cortisone without side effects or the need for hospitalization. Steffensen[2] reports the use of cortone acetate‡ (1 cc. cortone acetate to 4 cc. of normal saline) gave no deleterious effect nor irritative eye symptoms, and no corneal or conjunctival atrophy was produced.

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‡The cortisone used was the 11-dehydro-17 hydroxycorticosterone acetate of Merck & Co., Rahway, N. J. We acknowledge with gratitude the cooperation of Dr. J. M. Carlisle, Medical Director of the Merck Laboratories.

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In view of this we were confident that a similar saline suspension of the cortone acetate used locally in the nose would have no deleterious effect on the nasal mucosa and might relieve allergic nasal symptoms, and the results of the ophthalmological treatment suggested there would be no systemic effect.

Since in its current usage the term "allergic rhinitis" is rather loosely descriptive of a congested or stuffy nose due to a variety of causes, we were critical in our choice of cases, selecting 25 only after careful scrutiny of a detailed past history, and after a complete ear, nose and throat examination and nasal smears were made. Only two of this group were children; 17 were men, six women. Of these cases, all had had nasal symptoms for many years; in many the diagnosis of allergic rhinitis had been made, either by other physicians or by us; several had asthma; 16 had nasal polypi, others had had polypectomies previously; 17 had been found sensitive on allergic studies; and most had had allergic treatment in one form or another, with or without benefit.

The experimental work began in April, 1950, and while the duration of therapy varied in each case, all patients were followed for several months. In order to observe any effect the warmer weather, with its tendency toward lessened infection, might have in the relief of nasal symptoms, the cortisone therapy in the majority of cases was discontinued during July and August.

In the preliminary phases of the investigation each patient underwent a four-hour period of observation in the clinic, during which time the nose was sprayed every half hour and detailed examination followed each application. No general reactions were noted. All patients experienced relief of nasal stuffiness and postnasal drainage. They were re-examined after a 24-hour interval and all reported they were free of any irritation and had had no local or general symptoms. The first few patients so treated were then given a quantity of the solution with instructions to spray each side of the nose four times a day at home and return for further clinical observation. No ill effects were noted and each of the patients following in the series likewise used it at home. At each return visit the nose, sinuses, nasopharynx and pharynx were carefully examined. Nasal smears were routinely made.

Of the 25 treated, 12 (48 per cent) showed marked improvement; seven of these had had previous allergic studies with recurrence of symptoms, either because the specific therapy failed to produce relief or they had discontinued their allergic treatments; six (24 per cent) had some improvement, but were classified as doubtful; the balance (28 per cent) had no improvement and were considered as failures.

As we were largely dependent upon the subjective response to evaluate our therapy, our tabulation of the results may be open to criticism; however, we feel these results are not much different from those produced by specific allergy.

The following six cases we feel are representative of the series:

IMPROVED.

Case 1: E. J. Age nine, female, white. Case No 505491. In 1947, a history was given of many colds, recurrent, blocked-up ears with a mild conductive deafness. A T. & A in 1948 gave relief of the blocked-up ears with return to normal hearing In 1948 and 1949 there were recurrent colds and stuffy nose.

In April, 1950, she again complained of a stuffy nose, blocked-up ears and cough. Examination revealed congested nasal mucosa which shrank readily with a decongestant, profuse mucus discharge in both nostrils and edema of the Eustachian orifices.

Nasal smear, April 11, 1950, showed 17 per cent eosinophiles; April 12, 1950, there were eosinophiles 14 per cent. Specific therapy was given in the form of the cortisone spray every half hour for three hours. April 13, 1950, she felt improved, and had less nasal congestion. Examination revealed only slight mucus discharge, and no edema of the Eustachian orifices. No eosinophiles were found on nasal smear. The following day the child awoke with slight deafness and nasal stuffiness, both of which cleared during the day with the use of cortisone nasal spray. April 15, 1950, there was a minimal amount of mucus discharge in the nasopharynx, no edema of Eustachian orifices, no coughing or sneezing. Nasal smear showed no eosinophiles. April 22, 1950, the child was symptom-free. Examination revealed a clean nose and clear sinuses. On awakening in the morning there was no phlegm in the throat. No further therapy was given.

On May 5, 1950, she was still symptom-free, without cough or nasal stuffiness. May 25, 1950, she was still symptom-free and her father, a physician, thought the "results were marvelous." Slight nasal stuffiness and sneezing for two days were reported on June 8, 1950, but this cleared without therapy. June 23, 1950, there was again a slight nasal stuffiness which lasted two days. There were no accompanying ear symptoms. One nasal spray each day gave complete relief. The nasal smear showed 5 per cent eosinophiles. On nasal examination no noticeable congestion, discharge or atrophy were found.

Comment: This young girl with recurrent bouts of nasal stuffiness and clear nasal discharge, with elevation of eosinophile count, cleared with local cortisone spray. The father believes child has shown marked improvement. The recurring allergic nasal stuffiness might have cleared spontaneously, but one is impressed with the rapid disappearance of the nasal discharge and the absence of eosinophiles in the nasal smear following cortisone.

Case 2: Dr. M. J. Age 44, male, white. Case No. 412060. This patient had a history of repeated, frequent head colds, nasal congestion, nasal and postnasal discharge and chronic cough of many years' duration. Had considerable therapy and sinus irrigations on several occasions.

In 1944, the antra were dark on transillumination; mucus discharge was present in both nostrils and there was nasal congestion. X-rays disclosed thickening of mucous membranes of ethmoids and antra with a polyp in the left antrum.

Allergic studies showed four plus reaction to molds, house dust and bacteria; three plus to ragweed, timothy, orchard grass, June grass and red top.

Specific allergic therapy, hapamine injections, penicillin parenterally in courses gave no relief; slight relief was obtained with pyribenzamine. Smoking and occasional drinking were discontinued without change in symptomatology.

In May, 1950, he complained of continuous nasal congestion and marked postnasal discharge, requiring at least four handkerchiefs each day. Nasal mucosa was red and congested on examination but shrank readily with a decongestant. There was a profuse, clear mucoid discharge in both nostrils and nasopharynx. Nasal smear showed many polys and only a scattered eosinophile.

The cortisone nasal spray was begun, and he continued its use at home four times daily. One week later he reported a 50 per cent improvement, characterized by less nasal congestion and nasal discharge, requiring two instead of four handkerchiefs. The coughing was not only less frequent but also less severe.

In June the patient reported the good results persisted although the cough and symptoms recurred if the spray was discontinued. He was able to sleep through the night without coughing and thought his sense of smell might be returning.

He still felt improved in July. His cough was slight. He still required two handkerchiefs. Examination revealed a red, congested nasal mucosa, not greatly changed from the previous examination except for a somewhat lesser amount of discharge. The nasal smear showed 1 per cent eosinophiles Patient stated his symptoms continued to recur when the solution was discontinued and he noted that the longer the solution was used the longer the interval of relief when the spray was discontinued

Comment This physician with an allergic nose had had no benefit from any previous therapy The symptoms were definitely relieved by cortisone only to recur if the therapy was discontinued The question is raised as to how long the therapy can be safely continued and whether or not the relief will be maintained

Case 3 F P Age 58 male white Case No. 404353 In 1944 this man gave a history of sneezing nasal and postnasal discharge and asthma. Examination at that time revealed a nasal polyp and chronic infection in the right antrum. Allergy studies revealed a four plus reaction to duck feathers goose feathers rabbit hair giant ragweed timothy and orchard grass, three to four plus delayed reaction to house dust alternaria and to the bacterial proteins Specific allergic therapy with improvement was given from 1944 through 1950

He was seen on May 2 1950 in our clinic complaining of a stuffiness of the left ear Examination showed a fairly marked nasal congestion with numerous polypi in both nostrils particularly on the right. The right eardrum was dull retracted but with no inflammatory reaction Nasal smear showed many polymorphonuclear leucocytes but no eosinophiles.

The local cortisone therapy was begun on May 2 1950 He returned May 23 1950 at which time the polypi had almost completely disappeared The patient stated he was at that time more comfortable than he had been for two years The nasal cortisone spray was discontinued. On July 8 1950 he returned for treatment because of a recurrence of symptoms The nasal polypi were found to have returned to their original size The cortisone nasal spray was resumed By Aug 28 1950 the polypi had disappeared entirely On Oct. 3 1950 examination revealed a clean nose no polypi and clear sinuses No evidence of atrophy of the nasal mucosa could be detected The patient stated he had been using the cortisone nasal spray once daily he was very markedly relieved of his nasal symptoms and he was generally enthusiastic about the treatment.

Comment One is impressed with the fact that the nasal polypi disappeared and with the rapidity of their disappearance upon the use of the cortisone spray as well as by the fact that the polypi recurred when the spray was discontinued

DOUBTFUL IMPROVEMENT

Case 4 H. A. Age 46 male white Case No 247933 The history in this case dates back to childhood with recurrent attacks of sneezing nasal obstruction and nasal discharge Three years previously he had had nasal polypi removed and a submucous resection and antromeatal windows done. Following this there was complete relief of nasal symptoms until one month prior to admission to our clinic

When seen by us he complained of a head cold with marked right-sided nasal obstruction. Examination revealed the antromeatal windows to be patent. There were several small polypi bilaterally and one large polyp in the right nostril.

On May 23, 1950, cortisone nasal spray was begun, and continued daily. June 16, 1950, he stated he was breathing much more comfortably. On examination the polypi were found to be markedly decreasing in size. The therapy was continued. On June 30, 1950, he returned with a recurrence of the nasal symptoms and the polypi had returned to their original size. July 10, 1950, the polypi were removed under local anesthesia. The pathological diagnosis was "allergic nasal polypi." The patient failed to return for additional follow-up.

Comment: Although the patient was temporarily improved by the use of cortisone spray and his polypi showed some early tendency to decrease in size, the improvement was not maintained and the polyps returned to their original size even though the cortisone spray was continued.

Case 5: A. C. Age 37, male, white. Case No. 600298. This patient stated that since moving to Detroit, nine years previously, he had had sneezing, nasal stuffiness and nasal discharge. The nasal polypi had been removed two years and two and one-half years prior to this.

On March 4, 1950, he was treated by us for acute maxillary sinusitis and asthma, both of which responded to antibiotics and to sinus therapy which included sinus irrigations. April 17, 1950, examination revealed no evidence of sinus infection. There were several small polypi in both middle meati and a slight mucus discharge in both nostrils and in the nasopharynx. The patient was free of asthma but stated he had a tight feeling in his chest, more marked in the early morning. Nasal smear showed no eosinophiles. Allergic studies showed a three plus reaction to molds and bacteria.

On April 17, 1950, the cortisone nasal spray was begun, and he was treated every half hour until six treatments had been given. There was slight relief of his nasal symptoms and the patient thought his sense of smell had improved. April 21, 1950, he stated he had noted no change in his sense of smell, but the postnasal drainage was less and the tightness of his chest had been entirely relieved. April 28, 1950, he was symptom-free but again had had that morning a tight feeling in his chest. May 5, 1950, there was slight nasal stuffiness. The nasal smear was negative for eosinophiles.

He continued his therapy for six weeks with one to three-week intervals when no cortisone spray was used. He noted the spray gave him relief of the nasal symptoms, but that these recurred when the spray was discontinued. An "acute cold" contracted during this period was relieved in an hour by pyribenzamine 50 mgm. — the first time relief had been obtained with this drug.

Comment: This patient was given a weekly vaccine injection during this period, which he felt to be of doubtful benefit. His recurring nasal symptoms seemed to improve with cortisone, but we were unable to evaluate accurately the results of the therapy.

NO BENEFIT.

Case 6: C. W. Age 40, male, white. Case No. 386366. In this case nasal polypi had been removed several times in the past. In 1946, allergy studies showed a four and three plus reaction to corn, house dust, alternaria and bacteria. He was given specific allergic therapy and also hapamine injections with subjective improvement.

On Sept. 6, 1950, he came to our clinic with symptoms of sneezing, nasal obstruction and profuse rhinorrhea. Examination revealed a nose filled with polypi. The sinuses were clear on transillumination. The nasal smear was negative for eosinophiles.

He was given cortisone to be used four times a day. After a two-week trial without benefit he was unwilling to carry on with it any longer. Examination revealed no changes in the size of the polypi and at the patient's request they were removed No further treatment with cortisone was given.

Comment: The period of treatment may not have been adequate, although in other patients the symptoms were usually relieved in this length of time.

DISCUSSION.

Cortisone, in a 1:4 dilution, appears to produce a more or less temporary local effect, sustained in proportion to the length of treatment, and which may be as beneficial perhaps as allergic therapy. Objectively, the use of the solution lessened the nasal secretions and decreased the edema of the nasal mucosa, even in those patients who felt they had experienced little or no relief. Surprisingly enough, many patients seemed to experience relief of mild asthmatic symptoms, such as wheezing, cough and tightness of the chest, though severe asthma remained unaltered. Headaches of allergic origin may be relieved. Polypi may be shrunk, or may disappear; in the latter it is too early to determine whether or not they will recur and, if so, what the interval before recurrence will be. There would appear to be no correlation between the presence or absence of eosinophiles in the nasal smear and the favorable or unfavorable response to the cortisone.

Comment: It is our present impression that the use of cortisone nasal spray produces a temporary relief of the nasal symptoms of allergic rhinitis. To date, no untoward general or local effects have been noted. No nasal mucosal atrophy can be discerned.

We are continuing our experiment in the hope of gathering further data on the effects of this drug when used locally in the nose. It may prove to be an adjunct in the diagnosis of allergic rhinitis. We hope to learn whether or not the effects obtained are permanent, particularly where the eradication of nasal polypi is concerned.

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STENOSING PEPTIC ESOPHAGITIS, WITH A REPORT OF THE RESULTS OF TREATMENT IN 22 CASES *†

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Esophageal stricture due to nonspecific esophagitis is a relatively rare but important cause of esophageal obstruction. During the past 20 years, 22 cases of stenosis due to esophagitis were admitted to the Stanford University, San Francisco County and St. Joseph's Hospitals in San Francisco A study of the records shows that many of these patients were difficult problems in diagnosis and that treatment was often unsatisfactory.

Esophagitis in its mild form is the commonest disease of the esophagus and symptoms may consist of no more than heartburn or sour eructations,[12] however, as a complication of esophagitis there may occur a cicatricial stricture or a "peptic ulcer" of the esophagus, or both It is important to avoid classifying these complications as separate disease entities. The cases to be reported here all developed esophageal stenosis as a result of esophagitis. They are being considered together as a group primarily because they provided the esophagologist and the surgeon with a specific type of problem in the diagnosis and treatment of esophageal obstruction. Before presenting this series of cases in detail, the etiology of esophagitis will be discussed and the differential diagnosis and treatment of strictures secondary to esophagitis will be reviewed.

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ETIOLOGY.

The pathological process under examination here is characterized by a nonspecific inflammatory reaction which begins in the esophageal mucosa and may extend to involve all layers of the esophageal wall. Single or multiple ulcers sometimes occur and can penetrate or even perforate the muscular coats. As in our cases, esophagitis may lead to intramural fibrosis and stricture. Esophagitis and the strictures resulting from it are practically always found at the distal end of the esophagus just proximal to the cardioesophageal junction.

It is now generally accepted that esophagitis is the result of the corrosive action of acid gastric juice on the esophageal mucosa.[4, 10, 13, 15] Ferguson and his associates[22] have recently studied the effect of acid gastric juice on the esophageal, gastric and intestinal mucosa of animals and man. They found that the esophageal mucosa is especially vulnerable to damage by acid-peptic digestion and that gastric and intestinal mucosa are considerably more resistant.

Allison also makes the point that the mucous membrane of the esophagus has a low natural resistance to peptic digestion. He then calls attention to the important rôle of the mechanism at the cardia of the stomach which normally prevents gastric juice from flowing back into the esophagus—except to a limited degree.[35] Conditions which decrease or abolish the valve-like action at the gastric cardia will favor an increased reflux of stomach secretions into the esophagus. In some such cases, acid gastric juice may be expected to cause inflammation of the esophagus.

Actually, it does appear that esophagitis is almost always associated with circumstances which in one way or another reduce the effectiveness of the mechanism of the gastric cardia. In this category will be found hiatal herniae, in-lying gastric tubes, prolonged vomiting, comitose or moribund states. All of these are known to predispose to esophagitis.

The frequent coincidence of esophagitis and hiatal hernia of the short esophagus type deserves special comment. Short esophagus with a sliding hiatal hernia may be either congenital or acquired. In most instances, the abnormality is probably acquired.[3,33,34] Dey *et al.*[16] made some pertinent and interesting observations in dogs. By traction upon the liver or gall-bladder, by stretching the wall of the gall-bladder, cystic duct or stomach, or by stimulating the vagus nerve, they were able to cause such reflex shortening of the esophagus that the cardia of the stomach was pulled up into the mediastinum. A short esophagus with hiatal hernia was thus temporarily produced. It has been suggested that an irritant focus below the diaphragm in man (for example, chronic cholecystitis,[31] peptic ulcer or gastric cancer) may cause similar reflex shortening of the esophagus with hiatal hernia. Such an hiatal hernia would reduce the effectiveness of the mechanism of the cardia and permit reflux of gastric juice into the lower esophagus as a prelude to the development of esophagitis. Smithers[34] believes that in most cases seen by the radiologist, the shortened esophagus and hiatal hernia are due to contraction of the esophageal musculature and that normal relationships can be re-established by the relaxation of esophageal muscle spasm. In some instances, however, there may occur sufficient fibrosis from esophagitis to make permanent the esophageal shortening and hiatal hernia. Further progression of the esophagitis can lead to ulcer or stricture of the esophagus, or both.

Many patients with esophagitis have a peptic ulcer in the duodenum or stomach or give a history consistent with previous ulcer. In view of present concepts of the etiology of esophagitis, it is not surprising to learn that the peptic ulcer diathesis with its attendant hyperacidity, hypersecretion and vagotonia is a not uncommon background for esophagitis. Wangensteen and Leven[44] think that esophagitis is similar to duodenal ulcer in origin. They suggest that, next to duodenal ulcer, esophagitis should be considered the commonest manifestation of ulcer disease.

The triad of esophagitis, duodenal ulcer and hiatal hernia of the short esophagus type occurs clinically with striking frequency.[9] The development of these three conditions in the same patient must be more than a coincidence and serves as an example of the inter-relationships discussed in the foregoing paragraphs.

It has been noted that significant numbers of patients with strictures from esophagitis are alcoholics[9] (see Table 2). It is likely that a high alcoholic intake in some way results in an increased tendency to develop esophagitis.

For our purposes we will accept the following propositions:

1. Acid gastric juice is the irritant responsible for esophagitis.
2. The esophageal mucosa is unusually susceptible to acid-peptic digestion.
3. When the mechanism of the gastric cardia becomes ineffective, reflux of gastric juice into the lower esophagus increases and esophagitis may result.
4. Treatment should logically be directed toward reducing gastric acidity or keeping gastric juice out of the esophagus, or both.

DIFFERENTIAL DIAGNOSIS.

As stated above, esophagitis is not uncommon and generally causes minimal discomfort. The complications of esophagitis, however, produce more definite symptoms. When ulceration of the esophagus (usually described as "peptic ulcer of the esophagus") occurs, moderate epigastric or substernal pain and possibly some dysphagia due to esophageal spasm may be the chief complaints. Massive hemorrhage or chronic blood loss can take place and, rarely, the ulcer will perforate the esophagus with devastating results.

When esophagitis leads to stricture, the outstanding symptom is dysphagia. The development of swallowing difficulties is at times preceded for a variable period by heartburn, substernal distress or symptoms suggestive of peptic ulcer. The patient may indeed have a co-existing peptic ulcer in duodenum, stomach or esophagus. On the other hand, painless and progressive dysphagia can be the only complaint.

Stenosing esophagitis may occur at any age. It is very rare in infants and children in whom it is secondary to a congenitally shortened esophagus with hiatal hernia.[3] These strictures are most frequently seen in the older age group, the majority of patients being men over 50. Inability to eat satisfactorily usually results in weight loss which may be considerable. Painful dysphagia and hematemesis or melena suggest that both stricture and ulceration are present. There are no characteristic physical findings.

As a general rule, the clinician seeing the patient with stenosing peptic esophagitis for the first time will also have to consider in his differential diagnosis various other causes of distal esophageal obstruction. The most important of these are traumatic strictures (due most often to chemical burns), achalasia, and neoplasm of the esophagus or stomach.

The development of dysphagia following the ingestion of lye or other chemicals plus the radiological and endoscopic evidences of a benign esophageal stricture are usually enough to make the diagnosis obvious. There is little likelihood that strictures of this type will be confused with those due to esophagitis.

Achalasia of the esophagus (also known as idiopathic dilatation, megaesophagus and cardiospasm) is seen predominantly in women, occurs also at all ages and accounts for about 20 per cent of cases of dysphagia.[28] It is important to point out that achalasia and stenosing peptic esophagitis are not the same condition nor do they represent different stages of the same disease. In achalasia there is dilatation, hypertrophy and lengthening of the esophagus without organic obstruction. The distal 3 to 6 cm. of the esophagus are relatively narrowed and classically there is no fibrous stricture. It is true that inflammation may result from retained food. Also, it is of special interest that peptic esophagitis frequently develops in patients with achalasia after cardioplastic operations which make possible the reflux of gastric juice into the esophagus.[6,33] In stenosing esophagitis, the esophagus shows minimal or no dilatation and, instead of being lengthened, the esophagus is often shortened with an associated hiatal hernia.

TABLE 1.

OPERATIONS FOR STENOSING PEPTIC ESOPHAGITIS (EXCLUSIVE OF GASTROSTOMY) AS REPORTED IN THE MEDICAL LITERATURE.

Author	*Operation*	No. of *Cases*	*Result*	Comment
Eggers, 1935[19]	Resection, esophagus with construction, antethoracic esophagus	1	Good	Followed 16 years[20]
Clark and Adams, 1945[13]	Esophagogastrostomy	3	Good	Followed a few weeks to a few months
Allison, 1948[3]	Resection, esophagus with esophagojejunostomy	5	Good, 4	Duration follow-up not stated. One postoperative death.
Mason, 1948[29]	Plastic on esophageal stricture (Heineke-Mikulicz)	1	Good	Followed six months
Sweet, 1949[38]	Plastic on esophageal stricture (Heineke-Mikulicz)	6	Good, 4 Fair, 2	Duration of follow-up not stated
Wangensteen and Leven, 1949[44]	Partial gastrectomy	7	Good	Followed 10 months to 8½ years[43]
Mautz, 1946[30]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 3 years
Wyndham, 1947[46]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 3 months
Haight, 1948[24]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 4 years[1]

Dailey, 1948[15]	Resection, esophagus with Esophagogastrostomy	3	Good, 2	Two patients followed 2½ years. One post-op. death[15]
Baumann, 1948[9]	Resection, esophagus with Esophagogastrostomy	1	?	No follow-up reported
Keeley, 1948[27]	Resection, esophagus with Esophagogastrostomy	1	Good	Followed 1½ years
De Amesti and Chechilnitzky, 1948[17]	Resection, esophagus with Esophagogastrostomy	1	Fair	Followed 7 months
Sweet, 1949[38]	Resection, esophagus with Esophagogastrostomy	20	Good, 19	Duration of follow-up not stated One post-op death
Case Records, MGH, 1949[11]	Resection esophagus with Esophagogastrostomy	1	——	Post-operative death
Stephens, 1949[37]	Resection, esophagus with Esophagogastrostomy	1	Fair	Duration of follow-up not stated
Van Aken, 1950[40]	Resection, esophagus with Esophagogastrostomy	2	Good	Duration of follow-up not stated
Alves, 1950[3]	Resection, esophagus with Esophagogastrostomy	1	Good	Duration of follow-up not stated

Total number of resections with esophagogastrostomy for stenosing peptic esophagitis—34.
Postoperative deaths—3
Operative mortality—9%.

Results of treatment in 31 survivors of resection. Good in 28, or 90%
Fair in 2, or 6%
Result not stated in 1, or 4%.

TABLE 2.

FINDINGS ASSOCIATED WITH STENOSING PEPTIC ESOPHAGITIS IN 22 PATIENTS.

	Number of Patients	Per Cent
Peptic Ulcer in the Stricture	9	41
Esophagitis Without Demonstrable Ulcer	13	59
Hiatal Hernia (All Types)	13	59
Hiatal Hernia of Short Esophagus Type	9	41
Evidence of Peptic Ulcer in Duodenum (9) or Stomach (1)	10	45
Combination of: Esophageal Stricture, Hiatal Hernia of Short Esophagus Type, and Evidence of Duodenal or Gastric Ulcer	6	27
History of Heavy Alcohol Intake	7	32

TABLE 3.

RESULTS OF CONSERVATIVE TREATMENT IN 11 PATIENTS WITH STENOSING PEPTIC ESOPHAGITIS.

Result	No. of Cases	Comment
Good	3	Follow-up periods of one and one-half, four and seven years.
Poor	6	*Two* patients, followed two and seven years, require treatment intermittently and are incapacitated (both are alcoholic). *Three* patients continued to have symptoms until they died of intercurrent diseases in two, nine and 10 months. It is probable that the malnutrition resulting from their esophageal strictures was a contributing factor in their early deaths. *One* patient died of mediastinitis after an esophagoscopy.
Unknown	2	Asymptomatic a few weeks after initial treatment, but no later follow-up information obtainable.

TABLE 4.

RESULTS OF SURGICAL TREATMENT IN 11 PATIENTS WITH STENOSING PEPTIC ESOPHAGITIS.

Indications for Operation	Name	Age	Sex	Operation	Result	Comment
Failure of conservative measures: 5 cases	J.J.	62	M	Gastrostomy	Good	Followed 1½ years
	E. L.	57	M	Gastrostomy	Poor	Died of hemorrhage from esophageal ulcer 10 years after operation
	E. C.	68	M	Gastrostomy	Poor	Postoperative death
	P. N.	69	M	Transthoracic repair of hiatal hernia, short esophagus type	Poor	Steadily declined, dysphagia continued, died 13 months after operation
	J. M.	67	M	Gastrostomy, then plastic to esophageal stricture (Heineke Mikulicz), then left phrenic nerve interruption	Poor	Followed 6 years. Has persistent dysphagia, pain and repeated esophageal hemorrhages
Bleeding duodenal ulcer: 2 cases	H K.	49	M	Partial gastrectomy	Good	Required one bouginage postoperatively but is now 21 months since operation and eating normally
	A.K.	77	M	Partial gastrectomy	Poor	Postoperative death
Preoperative diagnosis of carcinoma of esophagus: 4 cases	F. H.	51	M	Transthoracic exploration of the esophagus with *bouginage* under direct vision	Poor	Failed steadily, dysphagia continued, died 9 months after operation
	J. M.	59	M	Transthoracic exploration of the esophagus with "excision of periesophageal scar tissue"	Poor	Followed 4 years. Still requires bouginage and has occasional esophageal hemorrhage
	H. S.	51	M	Plastic to esophageal stricture (*Heineke-Mikulicz*) and vagotomy	Poor	Followed 1 year. Recurrence of dysphagia and heartburn
	F. L.	36	M	Resection esophagus and esophagogastrostomy	Good	Symptom-free for 10 months. Patient died with acute alcoholism. Autopsy showed no evidence of re-

The most difficult and important problem is to distinguish stenosing peptic esophagitis from carcinoma of the distal esophagus or gastric cardia. There is abundant evidence in the literature[9,11,17,19,27,31] and in the cases to be reported here (see Table 4) that this has sometimes proven difficult. When a history of dysphagia over a period of years is obtained, this, of course, favors a benign stricture; however, the symptoms of stenosing esophagitis and obstructing cancer may be quite the same and consist merely of dysphagia for a few weeks or months plus weight loss. At the San Francisco County Hospital, cancer was the cause of dysphagia almost 15 times as often as esophagitis. The relative infrequency of stenosing esophagitis as compared with malignant obstruction was probably responsible in our series for a failure to consider seriously the diagnosis of esophagitis preoperatively in cases which, in retrospect, were quite typical.

Roentgenographic examination in stenosing peptic esophagitis will disclose a partial or complete obstruction at the distal end of the esophagus. Specifically, the esophagram usually shows:

1. A proximal esophagus of normal size or with only minimal dilatation;
2. A funnel-shaped narrowing of the distal esophagus (without shelving margins) leading into —
3. An area of organic stricture of variable length (usually several centimeters) in which there is distortion or destruction of the mucosal pattern and sometimes definite ulceration.

The esophagus will frequently be shortened and associated with a hiatal hernia of the sliding type so that the cardio-esophageal junction lies in the mediastinum, occasionally as high as the root of the lung (see Figs. 1-5). Hiatal hernia of another type may be found in some cases and examination of the stomach and duodenum may show signs of active or healed peptic ulceration. The degree of esophageal obstruction may be increased by an element of esophageal spasm.

Carcinoma of the distal esophagus or gastric cardia with infiltration of the esophageal wall usually produces a Roentgen picture of constriction which begins fairly abruptly, often with shelving margins and evidence of an irregular filling defect in the lumen of the esophagus. The esophagus above obstructing cancer may be dilated but usually is not. It should be kept clearly in mind that it is impossible for the Roentgenologist to rule out cancer with absolute certainty even though the esophagram may be typical of stenosing esophagitis. Not only has malignancy been known to develop in a stricture due presumably to esophagitis[9] but carcinoma is also occasionally associated with short esophagus and hiatal hernia.[16,39]

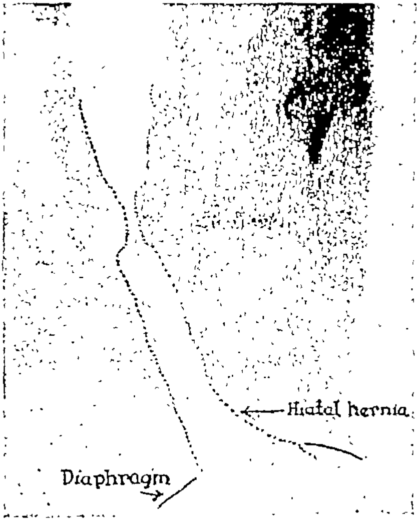

Fig. 1. Patient C. M., aged 67, male. Diagnosis: Stenosing peptic esophagitis. History: Dysphagia, intermittent, eight years. Chronic alcoholic. Treatment: Diet and repeated bouginage. Result: Poor. Followed two years.

Esophagoscopy is an essential diagnostic procedure in the usual case of stenosing esophagitis. Not only is it important to establish the diagnosis as a basis for therapy but the consequencies of overlooking resectable cancer are disastrous to the patient. While the gross appearance of the lesion at endoscopy may be indistinguishable from carcinoma, the changes seen in the esophagus are frequently quite typical. In obstruction due to esophagitis the mucous membrane often shows acute congestion for several centimeters above the stenosis, often with superficial discrete ulcers or extensive denudations. These acute superficial changes above the obstruction are important for they usually indicate a benign rather than a malignant lesion. Granulation tissue may be seen at the stricture which feels softer and more uniform

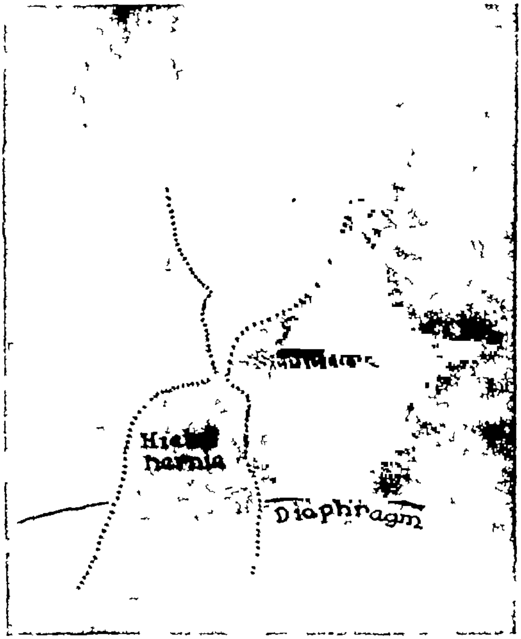

Fig 2 Patient F H, aged 51, male Diagnosis Stenosing peptic esophagitis History Dysphagia, one week Chronic alcoholic Treatment Exploratory thoracotomy with preoperative diagnosis of carcinoma. Since no neoplasm found at surgery, esophagus not removed. Result Died nine months postoperatively with continued severe dysphagia

than malignancy as a bougie is passed through it. When the Roentgenograms have been suggestive of cancer, the irregularities seen through the esophagoscope are found to be produced by extensive ulceration with granulation tissue.[3]

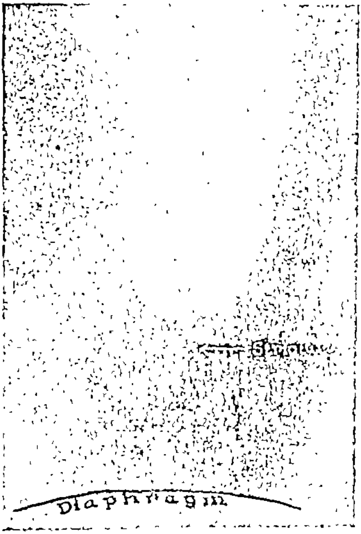

Fig. 3. Patient J. M., aged 67, male. Diagnosis: Stenosing peptic esophagitis. History: Dysphagia and hematemesis, three months. Treatment: Gastrostomy followed by transthoracic plastic to stricture (Heineke-Mikulicz), then left phrenic nerve interruption. Hiatal hernia and short esophagus found at operation. Result, Poor. Has persistent dysphagia and repeated esophageal hemorrhages. Followed six years.

Stephens[37] points out that the endoscopist can make the diagnosis of short esophagus and hiatal hernia when these are present by identifying the cardioesophageal junction well above the diaphragm. Biopsies immediately above the stricture will show esophageal mucosa, while those just below will contain gastric mucosa. In the last analysis, it is only by obtaining multiple negative biopsies that carcinoma can be ruled out with reasonable certainty. It has been emphasized that some biopsies should, if possible, be taken from deep within the stricture[9] and repeated endoscopies may be necessary. Finally, when a suspicion of cancer remains, an exploratory operation should be done.

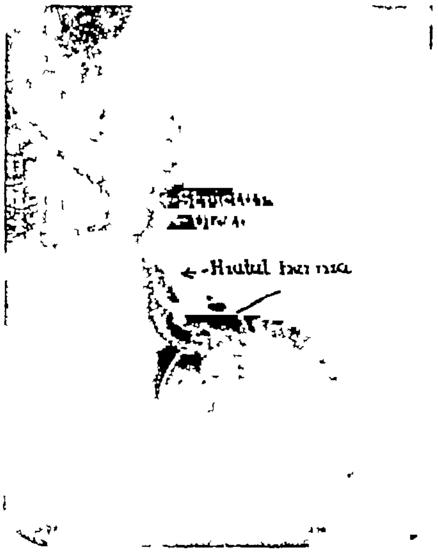

Fig 4 Patient H S, aged 51, male (Private patient of Dr Nelson J Howard) Diagnosis Stenosing peptic esophagitis History Dysphagia, four months Treatment Carcinoma of the esophagus suspected preoperatively Transthoracic plastic to stricture (Heineke-Mikulicz) plus vagotomy Result Poor Heartburn and some dysphagia persist Followed one year

TREATMENT.

Medical measures of value in esophagitis are bland, soft diet, frequent feedings, antispasmodics, antacids and mild sedation as for peptic ulcer. Elevation of the head of the bed to reduce the reflux of gastric juice into the esophagus while sleeping is advisable.

When a stricture prevents adequate food intake, endoscopic dilatation is required. It may be necessary to repeat this at varying intervals for many months as the stricture has a tendency to close down again gradually. Benedict and Sweet[9] found that bouginage and dietary treatment gave satisfactory results in over two-thirds of their 60 cases of stricture due to esophagitis. Certainly a thorough trial of this regimen is indicated.

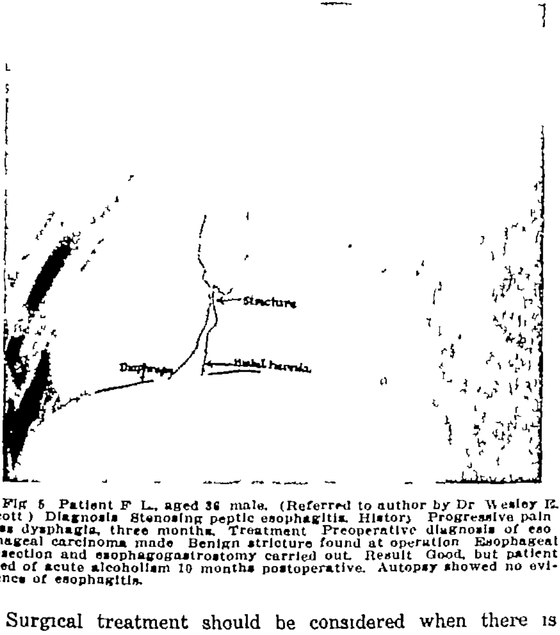

FIG 5 Patient F L., aged 36 male. (Referred to author by Dr Wesley E. Scott) Diagnosis Stenosing peptic esophagitis. History Progressive painless dysphagia, three months. Treatment Preoperative diagnosis of esophageal carcinoma made Benign stricture found at operation Esophageal resection and esophagogastrostomy carried out. Result Good, but patient died of acute alcoholism 10 months postoperative. Autopsy showed no evidence of esophagitis.

Surgical treatment should be considered when there is *1.* failure of repeated dilatations to relieve marked dysphagia, *2.* recurrent severe esophageal hemorrhage, *3.* intractable pain, or *4.* esophageal perforation Many of the patients with stenosing esophagitis are elderly and effective surgical measures are not without risk. Since the indications for surgery are to a certain extent relative, each case should have careful individualized evaluation before an operation is undertaken. There is no unanimity of opinion regarding the operation of choice for stricture due to esophagitis. A discussion of the various procedures which have been tried in this condition is, therefore, in order.

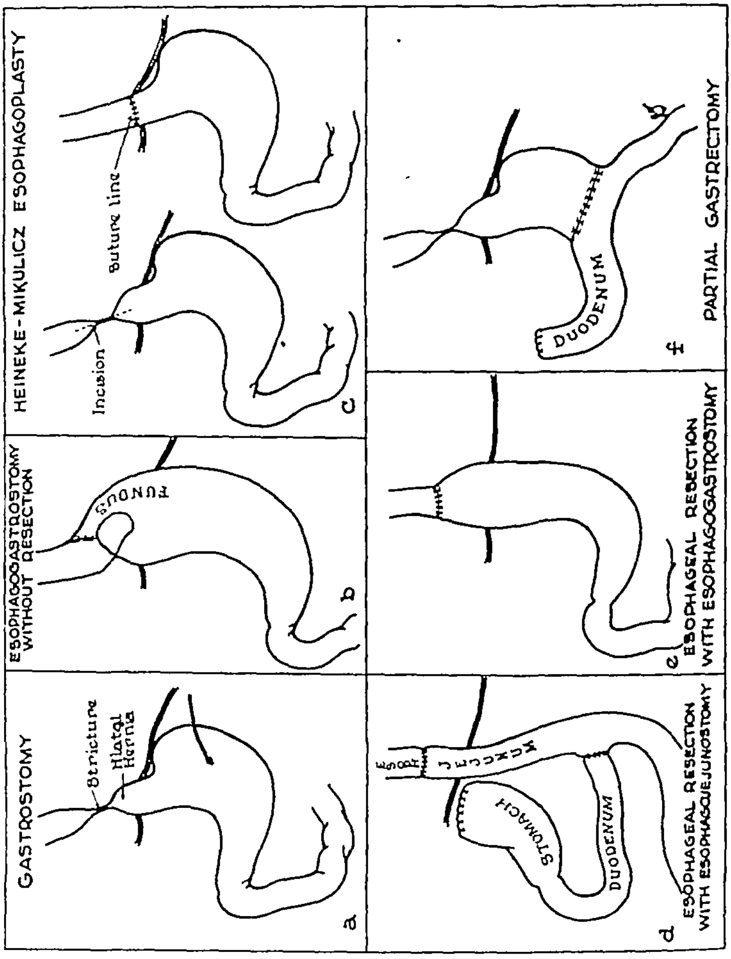
GASTROSTOMY
Stricture
Hiatal Hernia
a
ESOPHAGOGASTROSTOMY WITHOUT RESECTION
FUNDUS
b
HEINEKE-MIKULICZ ESOPHAGOPLASTY
Incision
Suture line
c
ESOPHAGEAL RESECTION WITH ESOPHAGOJEJUNOSTOMY
ESOPH.
JEJUNUM
STOMACH
DUODENUM
d
ESOPHAGEAL RESECTION WITH ESOPHAGOGASTROSTOMY
e
PARTIAL GASTRECTOMY
DUODENUM
f

Fig. 6. Operative procedures which have been used for stenosing peptic esophagitis. See text for discussion.

Gastrostomy: The simplest operation which has been used is gastrostomy (see Fig. 6a). This permits restoring the patient to better nutritional state but will not necessarily have any influence on the stricture.[31,44] It should now rarely, if ever, be necessary to resort to gastrostomy since parenteral alimentation is so effective, and the insertion of a small polyethylene tube into the stomach at endoscopy will permit adequate feeding for prolonged periods while conservative measures are being tried.

Esophagogastrostomy: Clark and Adams[13] have successfully employed an anastomosis of the fundus of the stomach to the esophagus above the level of the stricture (see Fig. 6b). This procedure will not prevent continued contact of acid gastric juice with the esophagus at the site of stricture and above it. It is conceivable that the inflammatory process in the esophagus might, therefore, extend upward to involve the region of the anastomosis. For this reason, the operation has theoretical disadvantages which do not recommend it for general use.

Esophagoplasty: Heineke-Mikulicz type of plastic procedure (see Fig. 6c) has been done by Sweet[33] in six patients who had short strictures. All patients were relieved of dysphagia, but two have complaints of heartburn and eructation of gastric contents. The result was very poor in the two cases in our series in which this operation was done (see Table 4). Both patients, one of whom had a vagotomy at the time of the plastic, developed heartburn postoperatively and dysphagia occurred. Since esophagoplasty neither reduces gastric acidity nor prevents reflux of gastric juice into the esophagus, it seems reasonable to predict that the long-term results will be unsatisfactory in a high percentage of cases treated in this manner.

Repair of Hiatal Hernia: It would seem logical to attempt to restore the competency of the mechanism of the cardia by repairing a hiatal hernia should one be present. When the stricture is not severe and the hernia is of the paraesophageal or sliding type *without* a short esophagus, this may be successful.[3] When there are extensive cicatricial changes and the esophagus is shortened by fibrosis, it is difficult to replace the esophagogastric junction below the diaphragm. Consequently, it is generally considered that hiatal herniae of the short esophagus type are not easy to repair satisfactorily.[23,31] Since most herniae associated with stenosing esophagitis are of the short esophagus type, repair of hiatal hernia has a limited usefulness but may be indicated in properly selected cases.

Partial Gastrectomy: Wangensteen and Leven[44] have reported good results in the treatment of seven cases of stenosing peptic esophagitis by a resection of three-fourths of the stomach as for duodenal ulcer (see Fig. 6f). They stress the importance of reducing gastric acidity and decreasing gastric emptying time. Certainly in their patients the response to gastrectomy was gratifying and supported their thesis that esophagitis is of acid-peptic origin.

Castleton and Dolowitz[12] noted the prompt healing of a bleeding peptic ulcer of the esophagus after a gastric resection. One of our patients with stenosing esophagitis was relieved of dysphagia following a partial gastrectomy for duodenal ulcer (see Table 4). On the other hand, stricture due to esophagitis has been seen to occur *after* a gastric resection for ulcer. Two such cases have been observed by Allison[2,4] and one by Van Aken.[40] The details of these resections with estimates of the amount of stomach removed and the postoperative levels of gastric acidity are not reported. One is, therefore, not entitled to infer that gastric resection will not give satisfactory results if done according to Wangensteen's specifications; nevertheless, these cases do suggest that the place of partial gastrectomy in the treatment of stenosing esophagitis requires further evaluation based on the study of a greater number of patients.

Esophageal Resection and Esophagogastrostomy (see Fig. 6e): This operation has been reported more frequently than any other in the treatment of these benign strictures (see Table 1). Sweet[38] has by far the greatest recorded personal experience with the surgical management of this disease. In 20 patients he did an esophageal resection and esophagogastrostomy with one postoperative death (due to coronary occlusion), an operative mortality of 4 per cent. The remaining patients have been relieved of dysphagia, although a few developed digestive discomforts. These are not described in detail nor is the duration of follow-up given. Sweet considers a wide resection of the stricture important in order to get above any inflammatory reaction in the esophageal wall which might dispose to recurrence of esophagitis and stricture.

Since good or fair results have been reported in 96 per cent of the 31 cases which survived resection and esophagogastrostomy (see Table 1), *it would seem that this procedure is better established than any other as a satisfactory operation* for stenosing peptic esophagitis.

Esophageal Resection and Esophagojejunostomy (see Fig. 6d): Allison[3] has considered the use of vagotomy in the treatment of peptic esophagitis but has rejected the procedure because he has seen peptic ulcer develop in the esophagus a few months after vagotomy for gastric ulcer; furthermore, vagotomy does nothing to alter the basic anatomical deformity which prevents proper function of the mechanism of the cardia and vagotomy will predispose to reflux by prolonging gastric emptying time. He has noted the development of ulcers in the esophagus in four of 21 patients following partial esophagogastrectomy with mediastinal anastomosis for carcinoma and these ulcers seemed to bear no relation to the acid secretion in what was left of the stomach. This has led him to stress the primary importance of the mechanism of the gastric cardia. He suggests that the best protection from a recurrence of esophagitis *is afforded by resection and esophagojejunostomy* rather than esophagogastrostomy. Allison has done a resection and esophagojejunostomy in five patients with one postoperative death. One died a year later of tuberculosis, but was apparently free of symptoms of esophagitis. The other three patients remain symptom-free, but the duration of follow-up was not given.

Esophagojejunostomy will indeed effectively prevent contact of gastric juice with esophageal mucosa and relief from esophagitis might be uniformly expected; however, a certain proportion of patients so treated is likely to develop serious digestive complaints as a result of the loss of the gastric reservoir and the passage of food from esophagus directly into the small intestine. More experience with this interesting procedure will be necessary before its possible undesirable side effects can be determined.

REPORT OF CASES.

In the 22 cases to be reported here, the diagnosis of stenosing peptic esophagitis was made by Roentgenography plus esophagoscopy (usually with biopsy) or by operation or autopsy. The stricture was just proximal to the cardioesophageal junction in all cases. There were 18 males and four females. They ranged in age from 36 to 81 years and 18 (or 82 per cent) were over 50 years of age.

The chief complaint of all patients was dysphagia. Gastrointestinal hemorrhage which varied from one-half cupful of fresh blood to massive losses occurred in eight patients (or 36 per cent). One patient died of hemorrhage from a peptic ulcer in his esophageal stricture.

The findings associated with stenosing esophagitis are shown in Table 2. The high incidence of hiatal hernia, short esophagus, peptic ulcer disease, and alcoholism is of interest and the implications of this have already been commented upon. Studies of gastric acidity were so frequently impossible or impracticable because of the esophageal obstruction that one can draw no conclusions from these cases on the relation of acidity to esophagitis.

Eleven patients were treated conservatively (see Table 3) and 11 were operated upon (see Table 4). A result was considered "good" if the patient was eventually relieved of symptoms, "fair" if he was improved but not asymptomatic, and "poor" if he was no better, worse, or relapsed promptly after temporary improvement.

Discussion: This series of cases is small and the high incidence of poor results has no statistical significance as regards the outlook for patients with stenosing esophagitis in general. It should be pointed out that all except two of these patients were from public clinic services. Many were first seen in a late stage of their disease with serious malnutrition and other complicating illnesses. Some had neither the resourcefulness nor the inclination to cope with their problem.

There were two other circumstances which influenced the results in the group of patients treated surgically. The first of these was the failure to make a correct preoperative diagnosis. In four of the 11 surgical cases, an incorrect diagnosis of carcinoma of the esophagus led to an exploratory operation which might have been deferred for a trial of conservative management. These cases were all sufficiently characteristic of stenosing peptic esophagitis to have stimulated further study had this condition been seriously considered. This limited experience emphasizes the fact that, although the clinical picture of stenosing esophagitis may closely resemble that of carcinoma, a careful differentiation is important in order to spare the patient unnecessary surgery.

Secondly, results in our surgically treated patients were influenced by an uncertainty on the part of the surgeons as to what constitutes an adequate operation for stenosing peptic esophagitis. This is not surprising since the condition is relatively rare and operative procedures are not standardized.

On the basis of the evidence so far reported, it seems reasonable to advise wide resection of the stricture and esophagogastrostomy as the most satisfactory operation for stenosing esophagitis. The vagotomy which accompanies this procedure plus the displacement of the fundus into the chest apparently combine to reduce gastric acidity and minimize reflux into the esophagus to such an extent that almost all patients so treated will remain symptom-free.

Finally, stenosing peptic esophagitis is a serious and sometimes fatal condition which is probably in many instances a manifestation of the peptic ulcer diathesis. Ulcer disease is notorious for its chronicity and tendency to remission and exacerbation. The results of surgical treatment of strictures due to esophagitis must, therefore, be accepted with caution unless based on follow-up of five or more years. Since most reports in the literature give only the early results of surgery, conclusions regarding the operation of choice are necessarily tentative.

SUMMARY.

1. Esophagitis is a common disease of the esophagus which may lead to ulceration, stricture, or both.

2. Reflux of acid gastric juice into the distal esophagus is the probable cause of esophagitis. The esophageal mucosa is especially vulnerable to acid-peptic digestion.

3. Hiatal herniae and other conditions which reduce the effectiveness of the mechanism of the cardia predispose to reflux of gastric juice into the esophagus and, therefore, to esophagitis.

4. When peptic esophagitis results in esophageal stenosis, bouginage plus medical measures as for peptic ulcer will give relief in most cases.

5. When conservative measures fail, esophageal resection with esophagogastrostomy is the operative procedure of choice.

6. Twenty-two cases of stenosing peptic esophagitis are reported and results of treatment discussed.

NOTE: The author acknowledges his appreciation to Nelson J. Howard, M.D., and to the University of California, Department of Surgery, at the San Francisco County Hospital for permission to include their patients in this survey.

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PHYSICIANS' ART SHOW

The American Physicians Art Association will have an art exhibit, as usual, during the A M A. Convention at Atlantic City, N. J., June 11 to 15, 1951, inclusive. Any physician in the United States, Canada and Hawaii desiring to participate in this show should communicate with the secretary for particulars.

J. Henry Helser & Co, Inc, Investment Managers with offices on the Pacific Coast, are the new sponsors of the American Physicians Art Association and will award 200 trophies besides a special Helser Trophy—a large decorative cup depicting Yankee ingenuity This cup is to be awarded for art work done in any medium Also, the large Popularity Trophy will be awarded to the owner of the art piece receiving the most popular votes during the A. M. A. Convention. Over 4,000 members of the American Physicians Art Association will receive shortly, entry blanks, shipping labels and rules about this fourteenth art exhibition.

The Annual Art Banquet will be held Tuesday evening, June 12, at the Marlborough-Blenheim Hotel, Atlantic City, N. J. Dr F. H. Redewill, Secretary, 720 Market Street, San Francisco 2, Calif.

SURGICAL PROBLEMS INVOLVED IN THE REMOVAL OF GLOMUS-JUGULARE TUMORS.*

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and
CHARLES S. LANE, JR., M.D. (by invitation),
Fort Smith, Ark.

In 1941, Guild[1] described the glomus jugulare (approximately 0.5 x 0.25 mm. in size), lying in the adventitial layer of the dome of the jugular bulb or along the tympanic branch of the IXth nerve in its bony canal, or rarely as it lies in the sulcus of the promontory. This structure resembled the normal carotid body histologically and was presumed to have functions like it. Four years later, Rosenwasser[2] reported the first recognized tumor of Guild's structure, and called the neoplasm a "carotid body tumor" of the middle ear and mastoid. During the past five years a substantial amount of literature has appeared on this subject. Lattes and Waltner,[3] in May, 1949, were able to find a total of 18 cases of their own and others, while in August, 1949, Lundgren[4] reviewed 16, of which he himself had observed four. These two lists overlap and diverge in such manner that they total at least 27 cases. DeLisa[5] added one case in 1950.

Winship *et al.*[6] reported a cervical lymph node metastasis in one patient, while Lattes and Waltner[3] found one liver metastasis; otherwise all of the tumors have been either benign or only locally invasive.

The terminology is confusing, in that Lundgren[4] prefers "tympanic body tumor"; Rosenwasser,[2] "carotid body-like

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tumor"; Lattes and Waltner,[3] "nonchromaffin paraganglioma of the middle ear," while we believe that the term glomus-jugulare tumor is the most informative.

Although this is our preference, the term "nonchromaffin paraganglioma" has much to recommend it. It will be recalled embryologically that the central nervous system is derived from the ectodermal neural tube, and that from the ganglion crest of this tube are differentiated the spinal ganglia. Some of the cells of the ganglion crest migrate ventrally to form the sympathetic ganglia; however, others instead of becoming neurons develop into gland cells, as in the adrenal medulla, and are aptly called paraganglionic tissue. They stain brown with chrome salts, whence the term "chromaffin." The carotid body, about the size of a rice grain, has long been considered paraganglionic tissue, and has histological similarity to a sympathetic ganglion The adrenal medulla and carotid body are two of many "pheochrome organs" scattered as paraganglia along the sympathetic nervous system. Watzka has been cited[3] as classifying the carotid body as nonchromaffin and nonepinephrine producing, yet paraganglionic; presumably the jugular body (glomus-jugulare) is in the same category. Guild[1] assumed the *function* of the glomus jugulare and carotid body to be similar. It will be recalled that the carotid body is an organ related to the reflex control of respiration and that its prime stimulus is anoxemia. It has nothing physiologically to do with carotid *sinus* reflexes regulating blood pressure.

In gross appearance, nothing is usually characteristic about glomus-jugulare tumors to distinguish them from ordinary inflammatory aural polyps, for which they are often mistaken. The fact that profuse hemorrhage may occur from the slightest trauma offers a clue to the correct diagnosis. In our Case 1 the Massachusetts General Hospital pathology laboratory rendered an interesting report regarding the gross pathology of the tumor examined there. "A rectangular segment of red-gray tissue measuring 1.7 x 1.0 x 0.8 cm. The material is spongy to palpation and is sectioned with fleshy resistance revealing red-gray moist cut surfaces from which escape large amounts of fluid blood. No vascular channels are visible grossly." The histological picture explains the bleeding tendency, since the tumor consists of a meshwork of blood spaces, most of which have a single layer of endothelial cells separating them from cords and nests of tumor cells, which resemble those of sympathetic ganglia. Mitoses have not been reported.

The average case will, therefore, seem to have an aural polyp, bleeding unduly, with chronic suppurative otitis media and mastoiditis. An attempt to state definitive clinical characteristics has been made by Lattes and Waltner[3] as follows:

1. An extreme tendency to bleed.
2. Pulsating tinnitus synchronous with the heartbeat.
3. Progressive locally expansive type of growth producing areas of destruction in the temporal bone.
4. Possibility of metastases.
5. Long duration of symptoms with repeated polypectomies and recurrences.

It is the purpose of this paper to describe the practical operative problems encountered in dealing surgically with glomus-jugulare tumors and to report three cases that have been observed at the Massachusetts Eye and Ear Infirmary. One of these, having such a tumor present itself in both the ear and nasopharynx simultaneously, is of particular interest, in that no similar case has thus far been described in the literature.

SURGICAL PROBLEMS.

The surgical difficulties encountered in dealing with this locally invasive neoplasm are chiefly the risk of hemorrhage; recurrence; possible metastasis; danger of facial paralysis from local invasion or surgical trauma; and inability to employ conventional anatomical landmarks in the operation.

event, the glomus jugulare location may be quite far removed from the hypotympanum. To compress the lateral sinus and tie the internal jugular vein would not control blood flow from the inferior petrosal, or the arterial supply of the bulb from the ascending pharyngeal artery; therefore, we feel that the bulb must be spared.

4. Facial paralysis or weakness is of frequent occurrence in glomus-jugulare tumors. Our Case 3 had had a complete peripheral VIIth nerve palsy for eight years prior to admission to the Infirmary. It had followed a secondary radical mastoidectomy. It seems likely from the history that an unrecognized glomus tumor was present then.

   In listing all available cases in the literature and their own as 18, Lattes and Waltner[3] found 10 to have facial paralysis or weakness, with six preoperative and four postoperative. Gentleness in dissection of the region of the VIIth nerve is important while removing the main tumor mass and may seem difficult in the presence of profuse bleeding.

   The very thin horizontal portion of the Fallopian canal between the geniculate ganglion and crest of the horizontal semicircular canal must be given special care as a prophylactic measure. A paralysis from local invasion of the nerve may be a part of the disease, but an earnest effort should be made to avoid it from simple surgical trauma.

5. It is our belief that inability to employ the usual anatomical landmarks, specifically the horizontal semicircular canal, in the operation may account for an occasional postoperative facial paralysis. Almost as good orientation can be obtained by creation of the tympanic spines (spina tympanica major and minor) when the bridge is removed. These spines lie external and anterior to the Fallopian canal, and provide adequate orientation until the main tumor mass is excised, thereby exposing the horizontal canal.

The technique described below was utilized in Case 1:

The classical Lempert endaural incisions were used, and adequate exposure of the mastoid cortex was obtained. A perforating electric burr was used to create a triangular-shaped opening in the mastoid bone in McEwen's triangle, the apex of the opening being directed toward the mastoid antrum. An oval cutting burr or a round semipolishing burr was then used to deepen this defect until the antrum was found.

The antrum was filled with very vascular tumor tissue from which free bleeding issued. This was controlled by exerting pressure with small gauze strips. The location of the horizontal semicircular canal, which would have followed as the next step in a conventional radical mastoidectomy, was deferred. The reason for this was that any manipulation of the tumor tissue increased the bleeding. A search for conventional landmarks by removing this vascular tissue was not feasible.

The principle then used was removal of bone with an electric driven burr from around the tumor tissue without touching the neoplasm itself. In order to accomplish this, the posterior-superior canal wall was thinned down and lowered, and the bridge was removed. The anterior and posterior tympanic spines were then visible. Using these structures as landmarks, the removal of the posterior-superior canal wall was continued and the epitympanum was widened.

Removal of this bone did not entail traumatizing any of the tumor. The neoplasm was kept free of the burr by gentle pressure with a small elevator. Bleeding was thus kept at a minimum.

Though the horizontal semicircular canal had not been seen, the approximate course of the horizontal portion as well as the beginning of the descending portion of the facial nerve could be determined. With the position of the nerve in mind, removal of bone necessary to open the middle ear widely was continued.

Because the inner portion of the canal wall epithelium appeared to be involved with tumor, this portion of skin was sacrificed. This was accomplished by cutting in circular fashion across the skin of the canal outside the limit of extent of tumor tissue and then separating the skin from the bony canal wall from without inward to the tympanic sulcus. Then the remainder of the bone work at the margin of the middle ear space and external auditory meatus was completed with the electric burr.

There now presented a sizable block of neoplasm from the outer margins of which bone had been removed. Very gentle separation of this tumor tissue from the bone that lay deep to it was accomplished with a small Lempert periosteal elevator. When sufficiently freed, it was possible by rapid dissection to remove *en bloc* the mass which had filled the mastoid antrum, middle ear, and inner portion of the external auditory meatus. Free bleeding was encountered as the block was removed, but this was controlled by firm pressure with gauze packing. A small amount of neoplastic tissue was left in the floor of the middle ear cavity where the tumor appeared to arise from the jugular bulb. As much residual neoplasm as possible was teased away until violent but easily controlled hemorrhage, apparently from the jugular bulb, was encountered. It was not of practical value to remove more tissue in this region. Other than for this, it was felt that complete excision of the tumor was accomplished. A complete radical mastoid cavity had been created; it was then finished completely.

Nine days later, a tumor clinic consultation was requested regarding possible irradiation of the hypotympanic region. The specialist from the X-ray department there made the following answer: "The histology of glomus tumor arising in the ear is essentially that of glomus tumors elsewhere in the body. They are notoriously radioresistant, and there is no reason to presume that those in this location should be more responsive. The literature is quite wanting in any enthusiastic statement regarding the value of X-ray treatments, although a number have been recorded. Treatment is not without hazard to the normal tissues in which the tumor lies, and I would not recommend this procedure. Even metastases have not been reported as having been favorably influenced by treating with X-ray."

On the seventeenth postoperative day, however, we decided to implant into the tympanum two radon seeds of one millicurie each. This was done under local anesthesia.

On the twenty-fifth day after operation the cavity was skin-grafted, and it healed uneventfully. Further details will be mentioned in the following case report.

CASE REPORTS.

Case 1: L. C, a 41-year old white female, was referred to the Massachusetts Eye and Ear Infirmary on July 5, 1949, by a private physician, with a diagnosis of polyp of the left middle ear

The history was as follows Eighteen years previously she had developed a slight hearing loss in her left ear which had not noticeably increased until the months immediately following the birth of her fourth child on Dec. 11, 1945. At this time she also noticed an increase in the quantity and frequency of a 'buzzing tinnitus which had occurred intermittently for several years

In May, 1946, while she was removing cerumen from her left ear with a hairpin, the hairpin "appeared to get caught" in the ear canal, and when she removed it there was slight bleeding from the canal for a few minutes. During the following week she developed a purulent, foul-smelling otorrhea with a definite increase in the hearing loss in this ear

In July, 1946, with the painless otorrhea still present, she first consulted a physician about the ear Following his initial examination, the otologist informed her that she had a polyp in her ear, and subsequently she was admitted to a hospital, where the polyp was removed.

From that time until December, 1948, she had no further otorrhea, but the hearing loss in the ear slowly progressed. At no time was there any pain, nausea, vomiting, vertigo or headache Occasionally she noticed a slight buzzing tinnitus localized in the left ear

In December, 1948, while she was scratching the external auditory canal of her left ear with her fingertip, she felt a soft mass. This caused her to consult her physician, who discovered another polyp in the canal, and she was once more admitted to a hospital where the polyp was removed.

There was no aural discharge from that time until March, 1949. At this time, however, a slight otorrhea developed, which progressively increased in quantity and became foul; also, she began to experience otalgia which was stabbing and fleeting in nature At no time did she have headache, vertigo, nausea or vomiting. Around the last of June, 1949, she again saw her physician, who told her that the polyp in her left ear had recurred. He then sent her to the Massachusetts Eye and Ear Infirmary.

The general physical examination was normal except for bilateral varicose veins of the legs.

Important local findings were limited to the left ear. Immediately inside the external auditory canal there presented the outer margin of a firm, grayish-white mass which completely occluded the canal. The canal was bathed with pus. The mass was slightly movable, and it appeared to be attached deep in the canal or in the middle ear. The auricle and mastoid were normal to inspection and palpation.

An air-conduction audiogram demonstrated normal hearing in the right ear and a 40 to 65 db loss in the left ear. The Weber test was lateralized to the left, and the Rinne test of the left ear was negative. Mastoid X-rays were reported: "Both mastoids are small. The left mastoid is increased in density."

Thirty cc. of iced alcohol syringed into the left external auditory meatus produced no nystagmus or vertigo. The fundi were normal; the neurological examination was not remarkable. X-rays of the base of the skull failed to demonstrate any bony erosion. X-rays of the hands, feet, pelvis and hips were negative for possible metastasis.

On July 12, 1949, a large portion of the mass presenting in the left external auditory meatus was removed with an aural snare. Profuse bleeding from the canal followed removal of this tissue, and required firm packing with gauze strips for adequate control. There was no bleeding when the packs were removed the following day. The pathological report of the biopsy was "glomus-jugulare tumor."

A left endaural radical mastoidectomy with removal of the tumor was then performed as previously described.

The patient's postoperative course was entirely uneventful. At the end of a month, she was ready for discharge. The cavity at this time had a very satisfactory configuration; the graft had taken well; the ear was dry. There had been no recurrence after a 13-month follow-up, and the cavity had remained dry.

Case 2: L. M., a 57-year-old white female, was first seen at the Massachusetts Eye and Ear Infirmary in September, 1944, complaining of discharge from the right ear for nine years. There had been an occasional attack of dizziness, but no true rotary vertigo, and there had been no headache, nausea or vomiting. She had noticed gradual progressive hearing loss in her right ear. She had also experienced several bouts of hemorrhage from the right ear which she had been able to control at home with cotton packs.

The general physical and neurological examinations were normal.

The local examination revealed the external auditory meatus of the right ear to be filled with a granular mass which bled when manipulated. Otherwise the local findings were not remarkable.

X-rays of the mastoids were reported as follows: "Both mastoids are small. The right mastoid is sclerosed; the left mastoid is pneumatized and clear."

On Sept. 19, 1944, the mass in the right external auditory meatus was partially removed by means of an aural snare. This procedure was accompanied by rather profuse hemorrhage from the canal, which was controlled by adrenalin packs. The hemorrhage recurred the following day, but the packing was removed the second postoperative day, without further bleeding. Biopsy report was "hemangioma."

The patient was discharged to the Out-Patient Department where she was subsequently followed. During the following month, the aural discharge ceased, and the remaining mass of tumor tissue deep in the external auditory meatus became covered with epithelium.

Caloric tests performed at this time demonstrated active labyrinthine reactions on both sides. The fistula test was negative.

The patient was not seen again until January, 1949. She then reported to the out-patient department complaining that her right ear had started discharging again several weeks after her last visit and had continued to discharge since that time. There had also been several bouts of slight bleeding from this ear, and three days before, she had experienced a rather severe hemorrhage which she had controlled with cotton packs.

She was readmitted to the Infirmary on Feb. 2, 1949.

General physical examination again was essentially negative. On local examination, the right external auditory meatus was found filled with a freely movable, smooth, grayish white polyp-like mass. The fistula test was negative. With the left ear masked, she had no hearing in the right ear.

Mastoid X-rays were reported: "The left mastoid is normal. The right mastoid has a few cells showing increased density. There is no bone destruction."

A biopsy was taken by removing a portion of the mass in the left external auditory meatus. Profuse hemorrhage resulted, which necessitated tight packing of the canal with gauze.

The pathological report of this biopsy was "glomus-jugulare tumor."

On March 8, 1949, a postaural radical mastoidectomy with removal of the tumor tissue was performed.

On March 16, 1949, the packing was removed with only slight bleeding, and the cavity appeared to be in good condition. On March 27, 1949, she was discharged and was subsequently followed in the Out-Patient Department.

Early in April, 1949, it was noted that the cavity appeared to be filling in with granulations, but the bowl was almost dry. Subsequently, the bowl became smaller and entirely lined with smooth epithelium. It was completely dry, and remained so when she was last seen, 22 months following her operation. There had been no recurrence observed.

Case 3: G. D., a 37-year-old white female, was first admitted to the Massachusetts Eye and Ear Infirmary on April 29, 1949, with a diagnosis of a mass in the nasopharynx.

Her previous history follows: In 1933, the patient developed a painless purulent discharge from her left ear. During the next several years, she had a progressive hearing loss in this ear, experienced recurrent exacerbations and remissions of the otorrhea, but she never had any pain, vertigo, nausea or vomiting. Treatment consisted of local cleansing of the external auditory meatus, the use of ear drops, and on several occasions "aural polypectomies" were performed.

On Aug. 14, 1940, the patient had a left postaural radical mastoidectomy. Following this operation, she continued to have left otorrhea. On July 1, 1941, there was a revision of the radical mastoidectomy following which the patient had a complete peripheral paralysis of the left facial nerve. The cavity became dry, but the facial paralysis persisted.

In 1946, she began to have bouts of mild, easily controlled epistaxis. The nasal obstruction was such that she was unable to get any air through the left side of her nose, and the right side was almost completely blocked also.

When she entered the hospital, her general physical examination was normal.

On local examination there was a complete peripheral left facial nerve paralysis. The plastic opening into the left radical mastoid cavity was markedly stenotic, and the cavity was moist. It contained a small amount of polyp-like tissue. There was a well-healed left postaural scar. Anterior rhinoscopy was normal, but examination of the nasopharynx revealed a firm, round, smooth, grayish-white mass completely filling the left half of this space. The mass appeared to have its origin from the superior part of the fossa of Rosenmüller.

X-rays of the nasopharynx and temporal bones were reported: "There is a mass in the left upper part of the nasopharynx which is very suggestive of malignancy. The right mastoid is normal. The left mastoid is postoperative. There is destruction of the lower part of the petrous bone —below the cochlea—which appears to be involved."

On April 30, 1949, a biopsy was taken from the lower limit of the mass in the nasopharynx, which included approximately one-fifth of the visible substance of the tumor. A profuse postnasal hemorrhage resulted, which required packing of the nasopharynx and left nasal cavity.

The pathological report on this tissue was "angioma, suggestive of glomus tumor"

Two days after the biopsy was taken, all packing was removed without further bleeding.

On May 3, 1949, the patient was discharged from the Infirmary to the Out-Patient tumor clinic. Here, during the next few weeks, a rapid resolution in the size of the mass was observed, so that by the end of May, 1949, the entire nasopharynx appeared free of tumor, and only a denuded area high in the fossa of Rosenmuller on the left side could be seen. She had experienced no further epistaxis, and she was breathing easily through both sides of her nose.

On June 20, 1949, the patient was readmitted to the Infirmary for investigation of the polypoid-like mass previously noted in the left mastoid cavity. A biopsy of this tissue on June 22 was reported as "glomus-jugulare tumor."

On June 27, 1949, an endaural revision of the left mastoid cavity with removal of the tumor was performed.

Pathological study of the specimen obtained from the middle ear region confirmed the original diagnosis of glomus-jugulare tumor.

The subsequent course of the patient is interesting. She was discharged two weeks after her operation. The mastoid cavity required about three months to become covered with epithelium; at first it seemed to fill with granulations, but then shrank after an epithelial covering grew in. It appeared to be shallow and stenotic, but various observers found no evidence of tumor recurrence in the ear up to and including the patient's last visit 18 months postoperative.

The nasopharynx remained free of any sign of neoplasm for nine months. Then a small purplish-red area of discoloration appeared at the orifice of the left Eustachian tube. This increased slightly in size during the next 11 months of observation, but the patient remained free of symptoms and no treatment was deemed necessary. She had gained 10 pounds since her operation.

SUMMARY.

Some of the practical surgical problems encountered in dealing with glomus-jugulare tumors are considered and three additional cases with this diagnosis are discussed.

One glomus-jugulare tumor presenting itself simultaneously in both the ear and nasopharynx has never been previously reported in the literature. In this case there was destruction of the petrous bone below the cochlea demonstrable on X-ray examination; an angiomatous neoplasm "suggestive of glomus-jugulare tumor" attached to the vault of the fossa of Rosenmüller on the side of a proven glomus-jugulare tumor in the mastoid. Anatomically, the extension of the lateral superior aspect of the nasopharyngeal wall as far as the anterior margin of the carotid foramen signifies with the above evidence that the "two" tumors were one and the same.

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RHINOGENIC BRAIN ABSCESS.*

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Rhinogenic brain abscesses have their origin about the nose, nasal cavities and paranasal sinuses, but brain abscesses originate from infected middle ears, mastoid, accessory nasal sinuses, the calvarium; from penetrating wounds or from metatases. As we are interested only in those abscesses which have their origin from a rhinogenic source, the discussion will be based on that premise.

ETIOLOGY.

The etiological factors are *1.* acute upper respiratory infections, especially those which produce acute purulent sinusitis; *2.* chronic sinusitis with an acute exacerbation, which becomes purulent with lack of drainage; *3.* extraction of teeth, which may produce a purulent maxillary sinusitis; *4.* fractures of the skull involving the sinuses; *5.* puncture wounds involving the nose, nasal cavities and sinuses; *6.* surgery in acute or green sinusitis producing osteomyelitis of the skull; and *7.* furunculosis involving the anterior nares or angle of the nose.

ROUTES OF INFECTION TO INVASION OF THE CRANIAL CONTENTS.

Invasion may be by continuity through bony defects consequent to osteomyelitis; local erosion of bone or fracture; through congenital defects in adjacent bony walls; by extension through preformed pathways; by way of blood vessels, especially veins; by way of perineural sheaths. The lymphatics may play a part in the routes of infection, according to some authors. Infection, which has reached the pia mater

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from an adjacent focus, usually gains entrance to the brain by way of an entering artery, an emerging vein, or the perivascular spaces Because of its more abundant blood supply the cortex does not yield to infection which extends into the white matter. The first stage of an abscess may be an infarct with arterial occlusion, hemorrhagic softening, or venous thrombosis The type of this primary lesion probably disposes to the type of abscess which is to develop As infection spreads in the decadent tissues, the clinical symptoms are those of encephalitis

BACTERIOLOGY

The causative organisms most frequently found in brain abscesses are as follows staphylococcus aureus hemolyticus, streptococcus hemolyticus pneumococcus, staphylococcus aureus, staphylococcus albus hemolyticus, streptococcus sub acidus, Gram negative bacilli, and streptococcus sub acidus hemolyticus. Anaerobic organisms do not favor the formation of a capsule in a brain abscess while aerobic organisms favor the formation of a capsule

PATHOLOGICAL FINDINGS.

Histological examination of brain abscesses reveals four zones of reaction, the inner zone of purulent material, the fibrous zone, the reactive zone of cellular structure and the encephalic edematous zone The inner zone is a necrotic zone composed of polymorphonuclear leucocytes, tissue debris and, in long standing abscesses, deposits of calcium This zone is surrounded by a reactive zone which is composed of loose connective tissue in which lie fibrin vessels and numerous cells, namely, polymorphonuclear leucocytes and lymphocytes The reactive zone is surrounded by a dense layer of fibrous tissue designated as a fibrous zone, and this is surrounded by a zone of polymorphonuclear cells in early stages with a few lymphocytes and plasma cells, and later with edema in the encephalic zone. Underlying the abscess is a large or small area of meningitis depending upon the size of the focus

Symptoms:

- General
  - Headache
  - Convulsions
  - Mental changes
  - Bradycardia
  - Vomiting
  - Dizziness

- Local
  - Palsy of contralateral VIIth nerve
  - Hemianoptic fields of vision
  - Aphasia
  - Papilledema
  - Hemiparesis
  - Orbital edema
  - Ocular motor palsy
  - Abducens palsy

ROENTGENOLOGICAL FINDINGS.

Roentgenological findings reveal changes in the osseous structure of the sinuses and the skull. Spinal fluid changes are not too great.

Diagnosis

- History of case
- Physical examination
  - General and local
- General symptoms
- Local symptoms
- Laboratory findings
  - Roentgenological
  - Spinal fluid
  - Bacteriological

Treatment

- Depends on what is found at examination
  - Expectant — medical
  - Surgical

Prognosis depends upon the status of the capsule formation. The increased intracranial pressure should be relieved by a decompressive osteoplastic flap with the excision of the abscess after a period of weeks when the capsule has become sufficiently strong to permit its complete removal without rupture. At present this method is thought to bring about good results in the way of a cure. Of course, if the abscess has been located, and drainage is established, the same results may be obtained as by decompression.

Heavy encapsulated abscesses have been carried for months without having been suspected of being brain abscesses, but it was thought at the time of operation that the surgeons were dealing with some other type of brain tumor.

1. How can one determine the status of the capsule?

a. By the length of involvement.

b. By diminution in signs and symptoms. Falling temperature, decreasing leucocytosis, stabilization of papilledema and an improvement of the general condition of the patient.

TREATMENT OF THE INFECTIOUS SOURCE OF THE ABSCESS.

Many cases improve under hypertonic salt solution for a period of time, but this cannot be continued for too long a period for fear of producing toxic dehydration. These cases later develop the same symptoms which they had at first, but during this period of improvement enough time has elapsed for the formation of a capsule.

In many cases of brain abscess the original source of infection has been eradicated or drained before coming to the neurologic surgeon. This was thought to be the better procedure, but recently some neurologic surgeons have thought it better to eradicate the abscessed cavity first, and the source of infection later. Personally, I would prefer the former method in most cases.

From a surgical standpoint if there is a definite capsule formation, the abscessed cavity should be removed as though it were a tumor; in fact, that is what it really becomes as far as symptomatology is concerned.

I am presenting a case of frontal lobe abscess which we first saw at Indiana University Hospitals in April, 1950.

B. M. R., a 30-year-old man, was admitted to the Robert Long Hospital, April 10, 1950, and was discharged May 25, 1950. History revealed that the patient became acutely ill about three weeks prior to admission following "postflu" when he began to have a swelling of the left eye, with pain above the left eye, in and around the orbit. This was accompanied by chills and fever. One week prior to admission the patient was placed on penicillin and developed a right hemiplegia.

Examination revealed a doughy supraorbital edema above the left eye with marked swelling and redness of the eyelids and orbit, so that the lids were entirely closed. There was paresis of the left oculomotor muscle. The pupils were small, equal with some irregularity on the left; no papilledema. The neck was stiff. He could not open his mouth well, at least not sufficiently to protrude his tongue. There was a right facial palsy with paresis of the right arm and right leg. Abdominal reflexes were absent. Examination of the nose revealed it to be full of pus. Reflexes on the right were hyperactive; Babinski was negative; there was no clonus. The patient was aphasic.

Lumbar puncture was done and opening pressure was 170, closing pressure 120; the fluid was clear and sparkling. Urinalysis was normal. Complete blood count showed hemoglobin 13, W.B.C. 10,350, bands 5, polys. 60, lymphocytes 27, monocytes 5, eosinophiles 2, basophiles 1. Spinal fluid showed red cells 30, white cells 11, globulin (Pandy) 2 plus, sugar 39, protein 124, serology negative, gold curve 3445555554. Spinal fluid culture revealed no growth.

X-ray studies showed normal chest. Films of the skull showed normal sella turcica, normal calcified pineal gland, no evidence of osteitis in the calvarium, petrous ridges and mastoid areas normal. The dorsal sella was normal. Films of the paranasal sinuses showed definite opacity of the anterior and posterior ethmoidal cells on the left and of the entire cavity of the left maxillary antrum with less translucency than that of the right. No bone destruction was detected. There was marked deviation of the cartilaginous portion of the nasal septum to the left. Loss of contrast over the frontal area was attributed to swelling of the scalp and forehead overlying this area.

Lateral films of the mastoids were normal.

On the day of admission the patient was placed under the care of Dr. R. J. McQuiston and the chief resident. He was taken to surgery and with incision through the left eyebrow in midportion of the orbit, the periosteum was elevated over the frontal sinus and a large subperiosteal abscess was encountered which exuded a large amount of pus. The orbital plate was found to be broken through and was removed. The cranial plate of the frontal sinus was examined and appeared in good condition. The opening was made through it 1.5 cm. from the orbital margin and good dura was seen. At this time a small bead of pus was seen to form and re-form at the upper outer margin of the frontal sinus. This opening was enlarged and was followed by a larger gush of pus estimated to be about an ounce. The orbital plate was found, removed and a brain abscess stalk was seen. A Pezzer catheter was passed over a stylet into the abscess cavity and the wound was sutured with silk. A left antral window was done using a rasp and antrum punches. Culture of the pus revealed strep. viridans.

Postoperatively the patient remained mentally clouded for several days, but gradually improved with improvement in his neurological picture with return of some function in his right-sided paresis. On April 18, 1950, a lipiodol injection through the catheter drain revealed an abscess of the frontal lobe area measuring approximately 2 by 2.5 by 3 cm. There was gradual improvement until April 27, 1950, 17 days after admission. At this time the aphasia became worse, the hemiplegia seemed to remain static. Visual fields at this time seemed normal. Neurological examination showed involvement of the left third with some resolution of the right seventh. Deep reflexes were hypoactive. No clonus was present; Babinski and Hoffman were negative. No sensory changes could be found.

Progress X-ray films indicated the cavity to be 4 by 5 cm., indicating an increase in size, and there was apparent sclerosis of the left frontal sinus in the frontal plate area which probably represented reactive bone to osteomyelitic involvement of the frontal plate.

On May 1, 1950, the patient was referred to the neurological staff for surgery which was carried out by Dr. Robert Heimberger and staff. A coronal incision over the frontal area was made, the periosteum was turned downward forward. A left bone flap was raised using five trephine holes and the dura was exposed. The dura was found to be densely adherent to the arachnoid over the area of the frontal lobe. Once the dura was incised and lifted, the brain was explored with a ventricular needle and the abscess was found in the frontal lobe. An incision was made in the cortex; this was carried downward and the lining of the cavity exposed. The entire lining of the abscess was then removed. The wound was thoroughly irrigated, the dura was sewn in place, the bone flap was replaced and kept in position by suturing the periosteum.

Pathological report was as follows Microsection: *a*. The lining of the brain abscess shows the features to be expected, *i.e.*, a central area of liquefaction necrosis with little or no architecture remaining. This is lined by a zone of fibroblastic activity and glial proliferation in which small numbers of polymorphonuclear leucocytes can be identified. There is also an increased vascularity of the wall with many thin-walled vascular channels in the thicker tissues at this site. In the central area of liquefaction necrosis numerous small basophilic bodies are encountered which are approximately the size of bacteria. Most of these appear coccoid. They may, however, represent tissue debris, but since both streptococcus viridans and staphylococcic epidermidis were isolated, these could well present bacterial colonies. *b*. The specimen from the cortex shows a congestion and edema. The meninges show the presence of a subacute or chronic meningitis with fibrous proliferation and increased vascularity as well as infiltrating cells, both lymphocytes and polymorphonuclear leucocytes.

Interpretation: This brain abscess is a typical picture and is associated with a meningitis as well.

Pathological and Bacteriological Diagnosis: Brain abscess (due to strep. viridans and staph. epidermidis, bacteriological cultures). Meningitis, subacute (presumably some etiological agent).

In the early morning following surgery, the patient suddenly went into a shock-like state, was cold, clammy with a pale color and drowsy. He was given a blood transfusion and improved.

By May 5, 1950, four days postoperatively, the patient was still aphasic but the wound was healing well. There was still some drainage from the sinus tract over the eye. The patient was rational, temperature was normal, he was eating and appeared much improved. By the time of discharge, May 25, 1950, there had been further improvement in his hemiparesis, the patient had begun to talk, the sinus tract had ceased draining and was healed and the patient seemed mentally competent.

On June 9, 1950, the patient was seen in the out-patient clinic. Two days prior to his visit he had suffered a Jacksonian type right-sided convulsion, following which he slept most of the rest of the day. Examination revealed right facial, hypoglossal paralysis which had almost disappeared. The patient's muscle power in the extremities was almost equal, there were no sensory changes, reflexes were slightly decreased on the right compared to the left. The patient was started on dilantin sodium and phenobarbital.

The patient was followed periodically in the out-patient clinic. The seizures occurred about one per month until in October when they increased in their frequency so that by November the seizures were occurring at about one per week, and the patient was suffering weakness and mental confusion accompanying these seizures.

The patient was readmitted Nov. 7, 1950, with discharge Nov. 18, 1950. Electroencephalogram showed S, with greater slowing at the left frontal temporal.

Physical examination on admission revealed a man with some difficulty of speech and inability to express himself. He seemed somewhat confused as to time and place. Neurological examination was negative except for some residual right, mild hemiparesis. Lumbar puncture showed clear spinal fluid and normal dynamics and normal chemistry.

Films of the paranasal sinuses showed the previous surgical defect, but no new developments. Pneumoencephalogram showed dilatation of the left lateral ventricle, especially of the anterior horn, suggesting herniation into the area previously occupied by the brain abscess. The ventricular system otherwise was normal.

While in the hospital on regular foods and medication, the patient had no seizures. Just prior to the last admission the patient had been quite a problem at home with temper tantrums and being easily upset.

The patient was last seen in the out-patient clinic Dec. 1, 1950. He had had only one convulsion since discharge from the hospital, and this had been accompanied by a marked increase in his hemiparesis which cleared up within a matter of hours thereafter. He had been somewhat less irritable but still became upset with the children; was able to help a little with housework. He was still aphasic but seemed somewhat improved and appeared to be primarily expressive. The left pupil was slightly larger than the right. Reflexes could be obtained more easily on the right than left but there was not much disparity.

The patient was continued on dilantin sodium and phenobarbital, and it was felt that he could probably be ready to attempt part time work soon.

SUMMARY.

1. Rhinogenic brain abscesses are rare.
2. I am quite sure antibiotics will further reduce their frequency.
3. The symptomatology is practically the same as other brain abscesses, especially if they are cerebral in type.
4. Encapsulation is necessary for recovery.
5. When well encapsulated, the abscess should be removed.
6. Aerobic organisms favor encapsulation of the abscess, and anaerobic organisms do not.
7. There is a question as to whether the source of infection should be eradicated first, or whether the abscess should be removed first.
8. Finally, a case is presented which presents all the symptoms which go with a brain abscess.

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FACIAL FRACTURES. CASE PRESENTATION.*

GLENN J. GREENWOOD, D.D.S., M.D.,
Los Angeles, Calif.

The mandible is a modified long bone,[1] sheathed in a reinforced mucoperiosteal coat. Fractures of this bone occur approximately four tmes oftener than those of the maxilla.[2] In contradistinction to other long bones, mandibular fractures tend to shatter. (This may, in part, be due to the snug mucoperiosteum exerting cortical pressure against the solidly contacted dental arch, thereby allowing for a minimum of cushioning of an exciting force.)

Two muscle groups and tendons control the mandible's function. The larger posterior group includes the temporal, masseter, the internal pterygoid and two-bellied external pterygoid muscles. The small anterior group lies mostly in the mouth floor and incorporates the mylohyoid, the geniohyoid, geniohyoglossus and the two-bellied diagastric muscles. Both muscle groups pull the mandible toward the midline, but the posterior group pulls upward while the anterior group pulls downward.

In this consideration of facial fractures, without loss of substance, local factors influencing displacement of fracture fragments (of the mandible and maxilla) are: the number, position and character of the teeth; the nature of the causative force; the bevel of the fracture lines, and the extent of soft tissue damage.

Because teeth serve as the principal structure for immobilization, mandibular fractures may be classified as:

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1. Teeth upon both sides of the fracture line. *2.* Teeth upon one side of the fracture line. *3.* Teeth absent upon both sides of the fracture line.[2]

Fixation of reduced mandibular fragments is accomplished by either the intraoral or the extraoral route, or a combination of both.

A. INTRAORALLY:

1. Wiring of the teeth is most universally used. It may be:

  a. Horizontal—that is, confined to one arch.

  b. Vertical or intermaxillary—that is, immobilizing the two arches in approximation. (Occlusion here being the ideal approximation.)

2. Utilization of an orthodontic band—arch appliance, or

3. Capping the teeth and soldering the caps to form a splint, and

4. Lastly, utilization of an acrylic splint.

B. EXTRAORALLY:

1. Roger Anderson's pin fixation.

2. Kirschner wire.

3. Head band appliance.

Simple fractures seen early should require only wiring. Compound fractures are relatively frequent. Extensive soft tissue damage may require constructing splints that prevent secondary contractures. This serves both esthetic and physiological purposes.

In fractures of the angle, wherein the ramus is depressed medially, *a convenient device to hold the ramus laterally is a* piece of shoehorn bent at an off right angle. This is fixed buccally to the intermaxillary wires, after the posterior flange has been adapted to the lingual surface of the ramus. This obviates external pin fixation. Many conservative operators use external pin fixation only when other measures are ineffective.

In children, interosseus wiring is advisable. Fine wire minimizes damage to the permanent tooth buds that occupy a large portion of the tooth-bearing segments.

Fractures of the maxillae are usually associated with those of other facial bones. Displacement, if present, occurs here, at the time of injury, because only the pterygoid muscles exert a substantial pull upon these bones.

Because of weak points that invite stress, fractures of this bone tend to follow definite patterns. Lefort recognized this in classifying three fracture types.[3]

Type 1: Bilateral maxillary fractures are frequently transverse. They pass through the tooth-bearing segment, including the palate, base of the nose and the medial and lateral orbital walls. Downward displacement may press upon the dorsum of the tongue. Upward displacement may impact into the antrum and fix the fragments between the malar bones.

Type 2: In the middle third fractures the line usually passes through the lateral antral walls, infraorbital canals, orbital floor, medial orbital walls and the nasal-frontal suture. This fragment, also, may displace upon the tongue or impact between the malar bones.

Type 3: The bones of the middle part of the face may here become detached from the skull, carrying along the maxilla's pyramidal process and its articulating zygoma.

Combinations of transverse and vertical fractures may be present. Treatment, to be adequate here, must restore facial contour as well as occlusion. Immobilization may be secured with intermaxillary wiring, retention splints, or plaster skull caps, the latter serving for anchoring of forked splints such as the Kingsley splint.[4] Impacted fractures may be mobilized with forceps. Cutting of adhesions facilitates moving older stabilized fragments.

The zygoma articulates with the frontal, sphenoid, temporal and maxillary bones. It forms the outer one-half of the orbital floor, enters into formation of the zygomatic and temporal fossa and gives attachment to the temporal and masseter muscles. This triangular shaped bone with its three processes helps buttress the maxilla on its lateral aspect.

Simple zygomatic fractures initially may be obscured by swelling, and deformity noted later, after the edema has subsided. Fragments when pushed medially and downward may rotate, thereby separating the frontal and sphenoid articulations. In the orbital cavity this may carry both the suspensory and palpebral ligaments inferiorly, stretch Tenon's capsule and result in diplopia in the first instance, and ptosis of the lateral canthus in the second instance.

Depressed fractures may ride upon the mandible's coronoid process, thereby preventing closure of the mouth.

Comminuted fractures may splinter the orbitoantral party wall, the fragments thus impinging upon either or both of these cavities. A hematoma may also complicate these fractures.

Treatment is facilitated by a local or light general anesthesia such as sodium pentothal. A combination of both may be used. Anteriorly placed depressed fractures may be elevated without incision by using simple traction with a hook or tenaculum; however, a more direct approach through a buccal vestibule incision facilitates easy reduction and permits great leverage to be exerted.

When the temporal bone's zygomatic process is also fractured along with the zygoma, the temporal muscle route affords the most direct approach for reduction. Cleaning out a hematoma or removing bony splinters or replacing and packing fractured walls may be most advantageously done through a radical maxillary antrum operation. Here counter drainage through an intranasal antrum window is advisable.

Open reduction with wiring, or introduction of a bone graft may be necessary. Especially is the former advantageous for maintaining the frontozygomatic contour with wiring.[3]

In the nose, the adult's nasal bones fuse in the midline. Hence, in contradistinction to fractures in children, fractures here are often bilateral. The Manhattan Eye and Ear Hospital in a series of 190 nasal fractures reports that 80 per cent occurred at the junction of the thin bony tip with the thickened body.[2] This point may be identical to the place where the upper border of the underlying upper lateral cartilage terminates cephalically.

Frontal forces sufficient to comminute the nasal bones may fracture the underlying septum. Lateral forces may cause one nasal bone to impact under the maxilla's frontal process, or the whole bony arch may at another time be swung free of all its articulations.

In the cartilaginous portion of the nose, the upper lateral cartilages' alae fuse with the septum. The lower lateral cartilages, however, are free and over-ride the upper lateral cartilages; hence injury, dislocation or fracture of the bony nose also involves the cartilaginous framework to a more or less degree.

Simple fractures seen early are easily repositioned under either a local or light general anesthesia. An elevator, swathed with cotton, moistened with saline and dipped in vaseline usually suffices for elevation. Impactions may require using an Ash or Walsham's forceps. The above procedures should suffice for contour restoration; however, attempts to realign the septum are more difficult. Especially is this true at the osteocartilaginous junctions, and particularly on the palatomaxillary floor. To satisfy best physiological results, submucosal replacement of the displaced osseocartilaginous tissues is often indicated. Stabilization of the displaced tissues is usually accomplished with vaseline strips, firm enough to maintain fragment position but loose enough to facilitate adequate circulation. Externally a cushioned stent dressing, anchored by adhesive, will maintain good immobilization for 24 to 48 hours. Comminuted fractures when not satisfactorily treated by the foregoing, may be suspended from a frontal head piece with a Kazanjian splint.

Complications of facial fractures are the exception. Rarely a basal skull fracture may be present. This may be noted by bleeding from an ear canal, but it is to be remembered that trauma from a condyle may also produce bleeding here. Cerebrospinal rhonorrhea may be proof of intracranial injury.

Primary arterial hemorrhage in the floor of the mouth is often troublesome until the offending artery, usually the lingual, is ligated. A hematoma may complicate a nasal fracture and is best evacuated early. Secondary orbital and lacrimal adhesions may occur, and require subsequent plastic procedures.

Malocclusion may require correction later. Infection, particularly with anaerobic organisms, used to be a dreaded complication, but chemotherapy and antibiotics have largely obviated it, with its sequela, osteomyelitis.

Delayed or nonunion, except in the aged, is usually of mechanical origin.

Although traumatic facial fractures are increasing, contemporary medical care is effecting a much better anatomical, physiological and cosmetic result. Too much credit for these good results cannot be given to our system of free enterprise in American medicine.

CASE PRESENTATION.

V. S., a 45-year-old white female was perfectly well until Oct. 26, 1950, when, between the hours of 12:00 and 1:00 P.M., while riding in an automobile she sustained head injuries rendering her unconscious. Emergency room examination revealed multiple contused facial wounds, a dislocated nose, a tongue that was slit back from the dorsum's tip for 5 cm. The nose was dislocated to the left, and a compound fracture was present in the body of the left mandible. Blood pressure was 160/85, pulse 88, fair quality, respirations 12 per minute and regular. Treatment for shock was instituted and a Barton bandage applied for mandibular immobilization. Subsequent history and physical examination were noncontributory.

Qualitative urinalysis was negative. The blood examination showed hemoglobin 11.7 gm. per 100 cc., red blood cells 4,010,000, leucocytes 7,250. The differential blood count showed polymorphonuclears 59, lymphocytes 40, monocytes 1.

Roentgenograms of the skull and chest were negative except for a fracture through the angle of the mandible on the left side. The anterior fragment was medially displaced and the posterior fragment over-rode it about 7 cm. After the patient gained consciousness and was out of shock, the soft tissues were cleansed, approximated and sutured, both intra- and extraorally.

Ten days later, after the orbital and liqual edema had subsided, because of difficulty in breathing through her nose, a rhinoplasty was done and the displaced upper and lower cartilaginous obstruction rectified.

Nine days later an unsuccessful attempt at a closed reduction of the mandible was attempted under local and light general anesthesia. An open reduction was then done through a left suprahyoid approach. The adhesions were severed, the fragments approximated and fixed with 22 gauge silver wire.

Because of an endentulous upper jaw an acrylic upper denture, previously made, was placed *in situ*. This acrylic denture had labial and buccal hooks to which the bands from the remaining lower teeth were attached, thereby completing intermaxillary immobilization. A Kingsley splint, which also had been previously affixed to the acrylic upper denture, permitted stabilization and fixation of the upper denture to a plaster headband, thereby immobilizing both jaws. This appliance was removed after six weeks, Roentgenograms showing a good callus formation.

This case was interesting for the following reasons: Initially the oral and glossal edema in the presence of nasal obstruction caused dyspnea, and prevented immediate immobilization of the fractured mandible. It was first necessary to permit the buccal edema to subside, then to restore the normal nasal airway, before reducing the fractured mandible. Delay in reducing the mandibular fracture permitted a fibrous union to form which made it necessary to use open reduction. Had the mandible been reduced earlier in the presence of nasal obstruction, it might have been necessary to resort to a tracheotomy.

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Audicon Models 400 and 415.

Manufacturer: National Earphone Co, Inc., 20-22 Shipman St., Newark 2, N. J.

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Manufacturer: Aurex Corp., 1117 N Franklin St., Chicago, Ill.

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Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

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Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.

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Silvertone Model P-15.

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Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

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Trimm Vacuum Tube No. 300.
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Vacolite Model J.
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Zenith Model 75; Zenith Miniature 75.
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PRESIDENT'S ADDRESS.*

LOUIS H. CLERF, M.D.,
Philadelphia, Pa.

One cannot accept the presidency of this Society without an appreciation of the great honor that has been conferred upon him and also a realization of his inadequacy. An understanding secretary and other officers have been helpful in solving the problems of the Society and I am grateful to them.

In a recent address before a group of physicians, General Paul R. Hawley, formerly Medical Director of the Veterans Administration and presently Executive Director of the American College of Surgeons, stated that, after having unusual opportunities for studying the professional activities of physicians in many States, he has regretfully arrived at the conclusion that the confidence of many people has been shaken regarding the unselfishness and the spirit of public service of the members of the medical profession. While we are not entirely unaware of this situation, we, nevertheless, are grateful to Dr. Hawley for his honesty in bringing this to our attention. His observations interested me very much and also suggested that physicians are not unlike the rest of mankind.

It has been generally observed that during the past quarter of this century there has developed a serious and continuing

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deterioration of former moral and ethical standards of behavior in nearly all classes of society in our own land as well as in other countries.

We have been and are living and working in an age which tends to accord undue importance to material interests and forget the urgent need for moral, ethical and spiritual considerations. We are continuously being confronted with the corrupting effects of political influence, intrigue and unethical practices upon our public servants in high as well as in low places. How does the medical profession fare?

No physician regardless of professional experience or position in the medical world can fail to recognize at this time the obligations owed by him to his profession. He should fully realize that he cannot consider himself an isolated individual in the medical world, but rather that he represents an important group and has the ability and opportunity to exercise a potent and an inspiring influence not only on the welfare of his profession but also in his community and his country. To exercise this influence, it obviously is necessary that we be fully aware of our responsibilities and our obligations.

In discussing the responsibilities and obligations of the physician, Dr. Elmer Hess, summarized them as follows: to alleviate suffering; to save life; to advise others of our methods; and, to secure compensation in order to live. I should like to add an additional obligation, namely, that the physician exert his individual influence in a more articulate manner to maintain the profession of medicine at a high ethical and moral level.

We are all in agreement regarding the alleviating of suffering. There should be no question concerning the saving of life. The physician should stay the hand of the Grim Reaper and not lend it aid. At this time the world is supersaturated with fears of war, of atomic bombs, and atomic thoughts, and the various "isms" are shaking the very foundations of our reverence for life. Until we learn more about life and death, it is unwise to consider making life shorter for those who are victims apparently of an incurable disease. We have learned too well of the terrible crimes perpetrated during World War II by members of our profession who contributed their knowledge and skill presumably in the interests of scientific research and used their brothers as experimental animals. This cannot be attributed entirely to the effects of a totalitarian form of government.

One of the fine things about our profession to which we can lay claim is our unselfish attitude towards a widespread dissemination of knowledge concerning methods and techniques to alleviate suffering and to save life. Our meeting today is an outgrowth of this desire to impart to our colleagues knowledge that we have accumulated concerning diagnosis and treatment.

It is generally recognized but perhaps too often ignored in practice that an important part of education, either academic, technical or professional, is the personal influence of the teacher upon the student There are many in this audience who are members of the teaching staffs of medical colleges. I wonder if that necessary inspiring influence of the personality and the ethical attitude towards patients is as clearly demonstrated by the teacher to the taught in medical colleges and hospitals now as it was formerly.

The laborer is worthy of his hire. The physician is entitled to secure adequate compensation so that he may live properly. The practice of medicine is considered by many as a trade and those who practice it as tradesmen. Some legal authorities have supported this thesis. This is a recent interpretation fostered by those who would deprive man of his dignity and confer it upon the State; who would usurp human rights and establish a socialistic regimen. It is no secret that there exists an impression on the part of the public, sometimes justified by factual evidence, that certain physicians seem to be a privileged and a not too ethical business group. To have saved a patient's life should not entitle the physician to exact ransom as does a kidnapper. This probably has served to undermine the confidence and esteem once enjoyed by our profession.

I shall always remember the words addressed by William Potter, former Ambassador to Italy, to the graduating class of one of our leading medical schools: "If there be within the sound of my voice any young man who has entered upon this task without consecration, without noble purpose and without high endeavor, and intends to continue in this heedless manner he had better leave our ranks at once and seek to obtain his chances for worldly success in some easier and more material channel!"

Regarding a more articulate attitude on matters pertaining to medicine, many of us have been ready and willing to allow those outside of our profession to solve these problems for us. Too many have failed to realize that a social revolution has been in progress in this country for several decades and that these changes are irreversible and irrevocable. If aware of it, they have not taken it seriously and have not yet felt the full impact of the changes that have already taken place. There can be little doubt that these will materially alter medical practice. Should we not take a more active part in these changes? If we leave it to the bureaucrats and politicians they will accept the credit for reducing the toll of disease and adding to the life span of man. They will urge us to give up a system which has prolonged human life by 20 years and to accept in its stead a plan which will make human life not worth prolonging.

As Fellows of this Society, we represent a select group who have attained a place of distinction in the field of otolaryngology. This includes many distinguished teachers. This places upon our shoulders additional obligations which we cannot delegate to others. There is need now, as never before, for a sound progressive medical leadership. Dr. Elmer L. Henderson, Past President of the American Medical Association, recently stated that "physicians by their thinking, spirit, and effort, can set an example for government, diplomats, and people everywhere."

The physicians' obligations are to care not only for the immediate ills of their fellowmen but also to aid in solving the social and economic problems involved on providing medi-

al care for citizens individually and collectively, and, in addi-
ion to exert their influence towards the development of good
vill among their neighbors across the back fence as well as
heir neighbors half-way round the world.

SYMPOSIUM: CARCINOMA OF THE LARYNX.

I — THE SURGICAL TREATMENT OF EARLY CARCINOMA OF THE LARYNX.*†

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The major consideration in the surgical treatment of early cancer of the larynx is selection of a method that offers the patient the greatest prospect of cure with the minimum amount of interference with the normal function of that organ. Of course, every laryngologist wishes he could see patients with intrinsic cancer of the larynx as early as it is humanly possible to discover them. This should not be difficult to attain since every case of cancer of the vocal cords makes its presence known by the appearance of vocal changes almost simultaneously with development of the lesion; nevertheless, approximately 30 per cent of patients with cancer of the larynx consulting the laryngologist for the first time have such extensive lesions that little can be done for them. Proper appreciation of the significance of persistent hoarseness by the physician, and by the laity, would reduce this incidence to a figure which would be practically negligible. Like cancer in other parts of the body, the earlier the diagnosis the greater the possibility of cure.

In the treatment of early cancer of the vocal cords, a complete cure is highly possible by two surgical approaches. Each of these is excellent for the type of case for which it was designed and successful results can be expected only if strict adherence to the indications for the use of each is observed. The first of these is intralaryngeal extirpation and the other

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is laryngofissure or thyrotomy. The relative indications, merits and results of these two surgical procedures will be briefly discussed.

INTRALARYNGEAL EXTIRPATION.

Experience has proved that in certain cases of cancer of the larynx an intralaryngeal operation can be performed with excellent results, provided certain fundamental requirements are rigidly observed. Occasionally, an early carcinoma is seen on one vocal cord which appears so limited in extent that one wonders whether an operation such as laryngofissure is necessary to eradicate so small a lesion. Such a growth can be successfully eradicated by the intralaryngeal approach, provided it is an early unilateral lesion, of low gradation and completely surrounded by normal appearing tissue. Such a small growth may well be considered a surface lesion. These small surface lesions are slow to infiltrate the surrounding tissue because of the usual low grade of malignancy and the paucity of the lymphatic supply in this area. The growth, which may be only the size of a grain of rice, a pea, or a dimpled match head, is usually located at the junction of the anterior and middle thirds of the vocal cord. If there is any question at all about the lesion being too extensive for intralaryngeal dissection, laryngofissure should be done. Because of the paucity of the lymphatics in the anterior portion of the larynx, the low grade of malignancy and the slow growth, these early surface lesions offer a greater percentage of cures at this stage of development than any other type of malignancy occurring in the larynx. This, then, is the ideal type of case for intralaryngeal extirpation; however, only 6 to 7 per cent of intrinsic cancers of the vocal cord are seen early enough to be suitable for this procedure. Leucoplakia and keratosis of the vocal cords, now being seen with greater frequency than formerly, are also indications for intralaryngeal extirpation. These lesions are precancerous and although repeated biopsies may show them to be benign, eventually they will become cancerous if not treated. For this reason we believe that intralaryngeal extirpation is indicated for leucoplakia of the cords of limited extent. To postpone treatment in order to watch developments of such growths is to invite trouble.

Successful extirpation of these highly selective lesions of the vocal cord is best accomplished by means of suspension laryngoscopy under anesthesia induced with sodium pentothal and curare. This method affords excellent direct visualization of the vocal cords and leaves both hands free to perform the operation. This approach is largely responsible for simplifying the intralaryngeal operation so that malignant lesions may be adequately treated.

After the larynx is exposed, the tumor is again carefully examined to check on its size and extent. The vocal cord is then retracted and its under surface examined. The tumor is grasped with fixation forceps and traction is made toward the median line, thus placing the cord on medial tension while the tumor together with adequate normal appearing tissue is excised. In accomplishing this we are ever mindful of the studies of New and Fletcher,[1] who investigated the microscopic extension of vocal cord tumors from the visible edge of the growth. After excision of the growth, the base is electrocoagulated and bleeding is controlled. Hospitalization is relatively short, there is minimal reaction, and postoperative tracheotomy has never been necessary in my series of cases. Vocal rest is observed and the patient kept abed for several days.

During the healing process the defect in the anterior half of the vocal cord is gradually repaired. Occasionally, granulation tissue develops, which, when persistent, may require removal and cauterization. Because of the small size of the tumor and the consequent limited portion of the cord removed, the wound heals rapidly. In many cases cicatricial tissue replaces the lost portion of the cord in such an excellent manner that it is impossible to recognize that an operative procedure had been performed on the cord. With such satisfactory repair, one can reasonably expect a voice far superior to that obtained in the average patient subjected to the laryngofissure operation. Patients who have had intralaryngeal extirpation of carcinoma of the vocal cord are kept under observation for at least five years, during which time the larynx is repeatedly examined

Again it must be emphasized that intralaryngeal extirpation of early carcinoma of the vocal cord can be successfully accomplished only in highly selective cases In a series of 49 cases there have been only three recurrences, or a curability rate of 93 per cent. This is the highest percentage of cures of any malignancies occurring in the larynx. These results are in no way remarkable because if the lesion is carefully selected it will be an early surface lesion, and theoretically all cancers would be curable if the diagnosis were made early and the proper treatment instituted immediately A small lesion on the vocal cord offers an opportunity for early diagnosis, and a higher percentage of cures will be obtained when advantage is taken of this opportunity.

LARYNGOFISSURE.

If early cancer of the vocal cord is found to be too extensive for intralaryngeal extirpation and yet not sufficiently advanced for laryngectomy, consideration must be given to the operation of laryngofissure, which is an ingenious technical procedure whereby the thyroid cartilage is split externally in the median line and its interior exposed to direct vision The indications for laryngofissure or thyrotomy are well established, although some elasticity must be permitted in interpretation of these indications because of the variations of the growth on the cord The ideal case is an intrinsic localized lesion occupying the middle third of one vocal cord with both ends of the cord free of any growth. Posterior extension with involvement of the vocal process and fixation of the cord is a definite contraindication to such a conservative operation Although ideal cases are seen with considerable frequency, more extensive lesions are sometimes encountered in which only by experience and judgment can the laryngologist determine whether the tumor is amenable to the laryngofissure operation Many growths extend to but do not involve the vocal process. Others extend to the anterior commissure and may even cross over to involve the anterior third of the opposite vocal cord. Leucoplakia involving a considerable portion of the vocal cord is being seen more frequently now than in former years. Repeated biopsies in these cases have been necessary in order to establish the presence of cancer and justify extirpation of one vocal cord. Incidentally, all patients seen by us with leucoplakia of the vocal cords have used tobacco excessively.

The technique of the laryngofissure operation is relatively simple and is usually performed under local anesthesia. An external incision is made on the median line of the neck extending from the hyoid bone to the level of the cricoid cartilage. Division of the thyrohyoid and cricothyroid membranes is necessary to facilitate exposure of the interior of the larynx. For division of the thyroid cartilage I have abandoned use of the electric saw because, regardless of the caution observed, bony necrosis occurred too frequently. I prefer the Clerf and Joseph hand saws and although more time is consumed in splitting the thyroid cartilage, no complications have developed. After the thyroid cartilage has been divided, the wings of the cartilage are spread open in order to expose the interior of the larynx to direct visualization. The vocal cords, ventricle and ventricular bands must be closely inspected and the supraglottic and subglottic regions must be examined for possible extension. Only after the growth on the vocal cord has been thoroughly studied in regard to its possible extension and depth can the operative procedure be formulated. The inner perichondrium of the involved side is dissected free and elevated from the inner surface of the thyroid cartilage well beyond the growth. The vocal cord with an adequate margin of normal appearing tissue on all sides of the malignant lesion is then excised. The bleeding areas are controlled and the entire base of the wound is electrocoagulated. The wound is closed tightly. As a rule we do not insert a tracheotomy tube unless dyspnea, secondary hemorrhage or a severe amount of emphysema develops. The safety tracheotomy through the cricothyroid membrane, as advocated by Schall,[3] has much merit and should be used more frequently. An Ace bandage is applied to the neck, and the patient requested to refrain from turning the head excessively. The patient is able to leave the hospital on the sixth or seventh postoperative day, although those living out of town remain available for observation for at least two weeks. Vocal rest is prescribed for a period of time.

Complications, although uncommon, do occur and necessitate constant vigilance. When a laryngofissure is done but tracheotomy is not performed, subcutaneous emphysema of the neck, face and upper portion of the chest may appear. This may be extremely alarming but it usually subsides within a few days after attacks of coughing have been controlled. Dyspnea seldom develops unless there has been excessive trauma and extensive surgical procedures have been performed. If secondary hemorrhage occurs, the wound should be reopened to control the bleeding, either with suture or a pack and tracheotomy.

In about 35 per cent of cases of laryngofissure, granulating tissue appears at the operative site during the healing process and at times this resembles recurrences. Although the surgeon is almost certain that the growth is granulating tissue, there is always an element of uncertainty and, on the part of the patient, much apprehension. If the granulations do not disappear, they should be removed by direct laryngoscopy and examined microscopically to determine whether or not there is a recurrence. If the presence of a recurrence is established, immediate action should be taken to eradicate as completely as possible the existing malignant growth. Although surgical treatment for recurrences is not especially successful, we have had patients in whom, following the laryngofissure operation, recurrences developed which necessitated laryngectomy and these patients have survived well beyond the usual five-year period.

If the vocal cord has been removed anterior to the vocal process, regeneration of the cord can be expected with good vocal function. In some cases excellent regeneration which compares favorably with the normal cord has occurred. If the vocal process has been partially removed, as becomes necessary at times, there occurs cicatricial fixation of the arytenoid resulting in altered vocal function. Occasional failure of the vocal cord to regenerate will leave a large empty space, which is responsible for a poor voice. In such conditions the voice can be improved considerably by resection of the thyroid alae on the affected side; this allows the soft tissue to collapse medially.

If the anterior third, or the anterior half of both cords has been removed, during healing considerable cicatrization, synechia and web formation in the anterior portion of the larynx may occur. Depending upon the extent of this process, considerable interference with breathing and speaking may result. Figi recommends the use of skin grafts in these types of operations with considerable success. The method suggested by McNaught[3] of division of the synechia intralaryngeally and insertion and fixation of a properly shaped tantalum plate through a small incision made externally through the thyroid cartilage has given excellent results. This tantalum plate is allowed to remain in place for at least six weeks. These patients are again happy because they note improvement in their voices and are able to breathe adequately. It might be advantageous to consider making this a routine procedure in cases in which the anterior portion of both vocal cords must be removed and excessive synechia and cicatricial tissue might be expected. I have had little experience with removal of a wedge-shaped section of thyroid cartilage at the anterior commissure. I am appreciative of Broyles'[4] excellent work in determining that fibres of the thyroarytenoid ligament extend into the substances of the thyroid cartilage.

The prognosis in properly selected cases for the laryngofissure operation is excellent, and if rigid adherence to the indications for ideal cases were observed, the percentage of good results probably would be much higher. Gradation of tumors is considered important and the higher the grade of the tumor, the greater is the possibility of recurrence and metastasis. Recurrences are seen in about 15 per cent of cases, which means that 85 per cent of these patients are cured by the laryngofissure operation. The laryngologist who first examines and operates on the patient has the best chance to effect a cure. His is the task of selecting the proper operation for the particular lesion. If his choice is correct, the operation will be successful and the patient will be permanently rid of the malignant growth.

In conclusion, intrinsic cancer of the vocal cords, if seen sufficiently early, will respond successfully to either the intralaryngeal or laryngofissure operation. Both of these are considered conservative surgical procedures by which a fatal disease can be completely eradicated with approximately 93 and 85 per cent cures, respectively, and vocal function, although damaged, is still preserved.

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SYMPOSIUM: CARCINOMA OF THE LARYNX.

II — TREATMENT OF EXTENSIVE CARCINOMA OF THE LARYNX.*

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Treatment of carcinoma of the larynx may be placed in three classifications: first, by laryngofissure; second, by total laryngectomy; and third, by some form of irradiation therapy. Dr. LeJeune has taken up the subject of carcinoma of the vocal cords. Irradiation therapy is the subject of the speaker following, and my presentation is the treatment of cancer beyond the scope of the vocal cords. In other words, the so-called extrinsic carcinoma of the larynx.

New, in his summation of the Symposium on Cancer of the Larynx before the Academy of Ophthalmology and Otolaryngology last Fall, suggested that I break down my extrinsic carcinomas of the larynx into their anatomical location. This I have done by re-examining all the specimens of cancer of the larynx that I have removed in my private practice. This paper will be the result of seeing, in private practice, 524 patients with carcinoma of the larynx, 59 of whom were inoperable at the time of their first examination. Seventy-nine refused operation, and 43 had some form of irradiation therapy, making a total of 181 cases. This leaves 344 patients who were operated upon.

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Of the 181 cases not operated, the distribution of the lesions were as follows:

Cords	57
Subglottic	12
Ventricles	7
Epiglottis	18
Epiglottis and tongue	16
Aryepiglottic fold	20
Arytenoid	14
Pyriform sinus and postcricoid	37
	181

It is difficult to believe, in the light of today's drive for early diagnosis, the large number of patients with true cordal cancer who refused operation Some of these may have later seen someone else and possibly have been salvaged; of these I have no further record. I should like to add that all of these were operable at the time of my examination. The hesitancy of the patient in having the operation at the time may, no doubt, have been due to some misguided advice of friends, or doctors, in saying that if your larynx is removed you cannot talk; such is not the case, as I have repeatedly shown. Any person can talk without his larynx if he makes up his mind to learn.

Of the 43 cases that had irradiation therapy, none are alive over five years.

In the other 59 cases that were inoperable, some form of palliative treatment was carried out, such as a tracheotomy.

The treatment of extensive carcinoma of the larynx means a total laryngectomy with the removal of sufficient tissue beyond it to assure adequate cure. If the lymph nodes are palpable these are to be removed at the time of operation with the larynx, beginning at the trapezius and working mesially to the larynx, removing all lymph-bearing structure, including the sternomastoid muscle, fascia, jugular vein, fat, and the lobe of the thyroid gland on the same side.

TABLE 1.
TOTAL NUMBER OF CASES OPERATED UPON, 344.

	Operated	Death Other Than Cancer Over 5 Yrs	Operative Death	Recurrence	Alive and Well	
					Over (5)	Under
Cords	132	23	2	6	69	32
Subglottic	62	4	0	17	26	15
Ventricle	38	5	1	10	16	6
Epiglottis	32	6	0	8	15	3
Epiglottis and tongue	21	8	0	7	5	1
Aryepiglottic fold	18	1	2	9	2	4
Arytenoid	17	4	0	1	6	6
Pyriform	24	2	1	13	4	4
	344	53	6	71	143	71

344 cases with six deaths = 1.7% OPERATIVE MORTALITY WITHIN THREE WEEKS OF OPERATION.

TABLE 2.
SHOWS THE PERCENTAGE OF FIVE-YEAR CURES ANATOMICALLY SITUATED, ALSO THE PERCENTAGE OF SUCCESSFUL CASES OPERATED UPON, MANY OF WHICH ARE NEAR THE FIVE-YEAR MARK WITH NO RECURRENCE.

	Operated	Over 5 Years	Under 5 Years Successful Cases
Cords	132	70%	93%
Subglottic	62	48%	72%
Ventricle	38	55%	71%
Epiglottis	32	65%	75%
Epiglottis and tongue	21	62%	66%
Aryepiglottic fold	18	16%	38%
Arytenoid	17	58%	94%
Pyriform	24	25%	41%

The larynx must be sacrificed in all of these cases, excepting possibly those cancers at the base of the tongue involving the epiglottis on the lingual side of a highly undifferential type, whereby suspension with diathermy as practiced by New has saved some of these cases over a five-year period.

CLASSIFICATION.

We must all realize that the site of the origin of cancer in the larynx is of the utmost importance. Anatomically it is of vast importance in deciding on the scope of the surgical treatment. It is true that only in the early stages can the true site of origin be definitely determined; in the more advanced cases by careful investigation into the history of the case, one may determine with a fair degree of accuracy where the original site of the cancer originated. This led to the first general classification by Krishaber, in 1879, of the intrinsic and the extrinsic group. The site of intrinsic cancer is quite definitely established. Much confusion, however, still exists as to

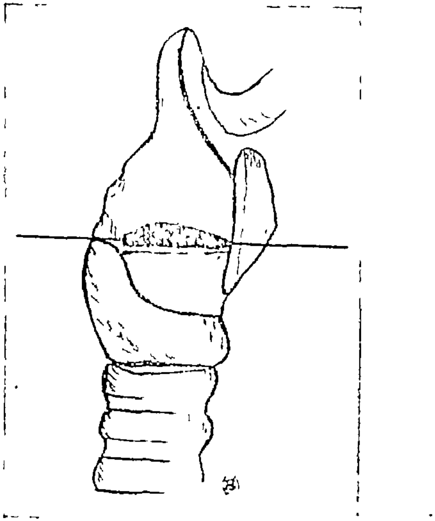

Fig. 1. Showing line drawn through ventricle, all below the line—intrinsic carcinoma of the larynx, all above the line—extrinsic carcinoma of the larynx.

the extrinsic group. Much clarification I am sure would result from an anatomical classification: cancer on the epiglottis, the arytenoid, the aryepiglottic fold and the pyriform sinus, to be termed the epilaryngeal type; the postcricoid and the lateral or posterior walls of the pharynx to be termed the hypopharyngeal type. This type of classification would establish the site of origin and present the surgical problems involved. Then the so-called mixed type, the result of an intrinsic cancer becoming extrinsic, or vice versa.

Intrinsic comprises lesions of the vocal cord which when seen early are amenable to laryngofissure; while in the subglottic area, laryngectomy is the procedure of choice.

EXTRINSIC OR EPILARYNGEAL.

This group of cancer of the larynx is causing a great deal of controversy as to treatment. It is true the degree of malignancy is higher than in the intrinsic type. Insidious in its onset, it spreads rapidly to the lymphatic nodes at a very early stage. Extrinsic cancer originates around the glottic opening, or on its outer surfaces. As I have stated above, this group should be subdivided into those neoplasms arising from the epiglottis; the aryepiglottic fold; the arytenoid and the pyriform sinus, the epilaryngeal group.

Cancer of the postcricoid area, the lateral and posterior walls of the pharynx to be known as the *hypopharyngeal* group. This classification definitely establishes anatomically the site of growth.

Cancer in this region is usually of the squamous cell type, although it may be papillary or occasionally of basal cell type. Sarcoma is also found. The lymphatic nodes are invaded early and consistently; the course is fairly rapid, and unless prompt treatment is instituted, a year and one-half is the average span of life for the patient. Then, too, in these cases where the disease has extended beyond the confines of the larynx and invaded some of the adjacent structures, a more extensive operation is indicated, such as laryngopharyngectomy.

LYMPHATICS.

The submaxillary lymph nodes drain the side of the tongue along with other parts of the face; therefore, with any involvement of the posterior part of the tongue from the lingual surface of the epiglottis, the submaxillary gland with its nodes should be removed.

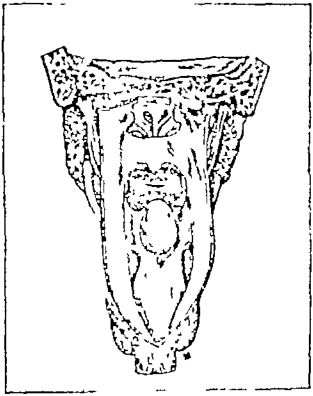

Fig. 1. Posterior view of pharynx showing areas in relation to extrinsic carcinoma of the larynx.

DEEP SET OF CERVICAL LYMPHATIC NODES.

A. The infrahyoid nodes. These lie on the thyrohyoid membrane and drain the front of the larynx. It is important to remove the pre-epiglottic space in laryngectomy.

B. The prelaryngeal lymph nodes. These lie on the cricothyroid membrane draining the larynx. Trotter has shown that their afferents pass through a small foramen in the middle of the cricothyroid membrane. These lymph nodes are often the first to become enlarged in cancer of the larynx. They also assist in drainage of the thyroid gland. It is well, therefore, to remove the cricoid cartilage with the larynx.

The deep cervical chain receives ultimately all the lymph from the sections above mentioned. This deep cervical chain consists of a number of large lymph nodes lying in relation to the cartoid sheath. A few of these occupy an outlying position behind the pharynx. This deep chain lies along the side of the pharynx, trachea and esophagus and extends from the base of the skull to the root of the neck. These are divided into the superior and inferior by the point of bifurcation of the common carotid, or the crossing of the omohyoid muscle; both are in close relationship with the internal jugular vein, and in removing them it is necessary to strip them from the vein, or better, the jugular vein should be removed with them. Some of the lymph nodes of the inferior group project beyond the posterior border of the sternomastoid muscle into the posterior triangle of the neck. There are a few nodes of this group that lie in the groove between the trachea and esophagus: they drain the thyroid paratracheal glands. A large lymph node, situated below the angle of the jaw between the internal jugular and facial veins is known as the main lymph node of the tonsil. Another large node at the bifurcation of the carotid just below the cornua of the hyoid bone is called the main lymph node of the tongue. Finally, a large lymph node on the carotid where the anterior belly of the omohyoid muscle crosses, it is known as the supraomohyoid lymph node.

The main lymph node of the tongue, in cancer of the larynx involving the base of the tongue, is the one that enlarges so rapidly postoperatively, and in many cases this lymph node may be removed and followed by irradiation.

The dissection of the neck should include the area from the angle of the jaw and tip of the mastoid downwards to below the posterior belly of the omohyoid muscle, removing the jugular vein, submaxillary gland and its nodes, fascia, the hyoid bone with the infrahyoid muscles and larynx *en masse*, with part of the thyroid gland if needed.

We must remember that the flow of lymph following a resection may have a retrograde movement, and involve the lymph nodes in the postauricular area, posterior triangle of the neck, ending in the retropharyngeal lymph nodes.

With involvement of the base of the tongue from the lingual portion of the epiglottis, we must remember that the cancer cells in the tongue may pass readily from one side to the other. From this it becomes necessary to deal with both sides of the neck. When we have a patient who has the entire length of the deep cervical chain of lymphatic nodes involved in a fixed hard, firm mass, extending to a point below the infraomohyoid muscle, it is needless to say that we cannot

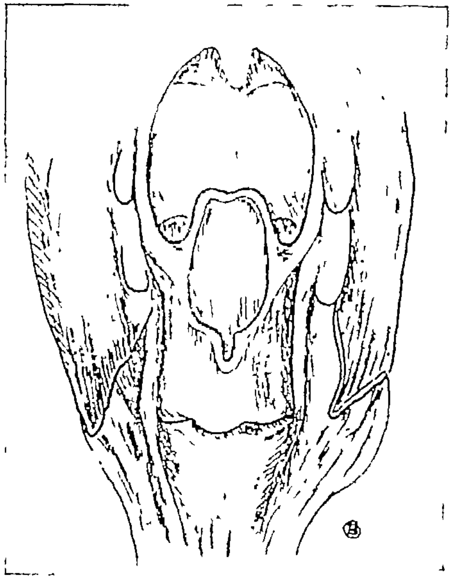

Fig 3 Showing areas involved in extensive carcinoma of the larynx.

expect to cure the cancer. By that time the cells have already entered the thoracic duct into the general blood stream.

Complete destruction of all cancer cells is at present the only known method of effecting a permanent cure, whether this be by surgery, irradiation or by some biological change in our makeup.

With this in mind, the surgeon when operating should be careful about disturbing the growth at the time of operation so as not to cause metastasis by inoculation. The operation should be sufficiently radical to get well beyond the growth, keeping in mind that New's observation on the microscopic extension of the growth taken at 5, 10 and 15 mm. from the apparent margin of the growth found the maximal extension of highly malignant tumors was 15 mm. This has been confirmed by Broyles and Snitsman, who have shown that the cancer cells have extended beyond the apparent growth histologically, in the anterior commisural growths of the cords, another reason why the pre-epiglottic space is invaded early and should be removed with the larynx. I have also demonstrated this at the time of operation in epiglottic cancer below the hyoepiglottic ligament.

PRE-EPIGLOTTIC SPACE.

Jean Leroux Robert published a paper, Les Epitheliomes Endolarynges, in 1936, in which he demonstrated the invasion of the pre-epiglottic space, and Tambien demonstrated histologically that cancer of the vestibule and ventricle of Morgagni invaded the pre-epiglottic space and stated that the classical laryngectomy is insufficient for the complete eradication of cancer in these cases.

It is true that for years I had been removing the hyoid bone in complete laryngectomy, but it was not until early in 1939 when I received from Dr. Ricardo H. Bisi, of Buenos Aires, his book on Cancer of the Larynx, published in 1938, that I realized that the hyoid bone should be removed *en masse* and not separately. I was shaving the base of this pre-epiglottic space instead of going completely around it. Since then I have recognized its value and have been following his procedure in all my laryngectomies and have not regretted it, as the results have been very satisfactory and gratifying.

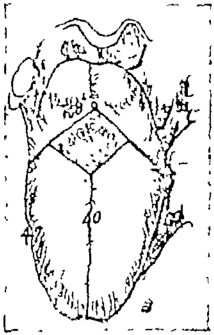

Fig. 4. Showing the relationship of base of tongue with epiglottis.

I feel that it is a most important step in the treatment of cancer of the larynx.

A true intrinsic cordal cancer limited to the midthird of one cord is ideal for laryngofissure, but with a growth at the anterior commissure you must go through the pre-epiglottic space, a very potential danger area for recurrence. We cannot be conservative when dealing with cancer. I agree with Clerf and thank him for bringing the subject up again in his paper, Pre-Epiglottic Space, in its relation to cancer of the epiglottis. His keynote was "that we should get away from conservative treatment of cancer of the larynx, we gain nothing by appeasing cancer by subperichondrial resection, narrow fields, etc."

This space is described by Robert as follows, "This space is triangular or funnel-shaped, in the sagittal section it is triangular with its base upwards and the vertex point downwards. It is an actual space filled with cellular and adipose tissue. It contains no lymph nodes. The anterior and lateral walls are composed above by the thyrohyoid membrane, and below by the anterior superior angle of the thyroid alae. The posterior wall is formed by the epiglottis from its middle to lower extremity, which is situated immediately above the

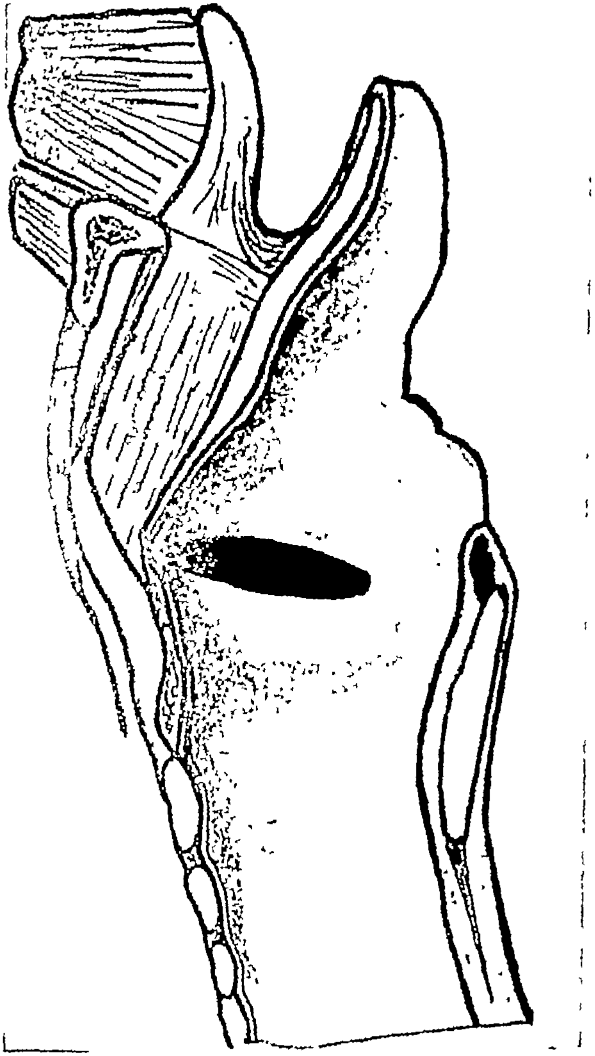

Fig. 5 Drawing showing the pre-epiglottic space.

anterior commissure of the vocal cord. It is closed above by an aponeurosis which forms the roof, and extends from the posterior surface of the hyoid bone and inner border of the cornua to the middle of the anterior surface of the epiglottis; the hyoepiglottic ligament. This space is often invaded by cancer which attacks the lower portion of the epiglottis, vestibule of the larynx and the false cord. In order to completely encircle these growths the entire larynx, pre-epiglottic space and the hyoid bone should be removed *en masse*."

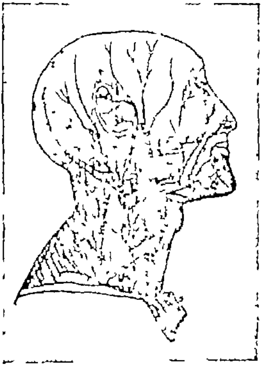

Fig. 6. Showing the lymphatic drainage in the neck.

It is common to find cancer cells microscopically in the pre-epiglottic space well beyond the apparent anatomical margins of the growth. This invasion cannot be determined beforehand, either by direct or indirect examination of the larynx. The pre-epiglolaryngectomy as designed by Bisi should be practiced in all cases.

LARYNGECTOMY.

The indications for laryngestomy are as follows:

1. When the growth has extended to the anterior commissure or has extended backwards to the arytenoid.

2. When the epiglottis is involved below the level of the hypoglottic ligament.
3. When the growth has crossed to the opposite side, and there is subglottic extension.
4. The growth springing from the ventricle or ventricular fold.

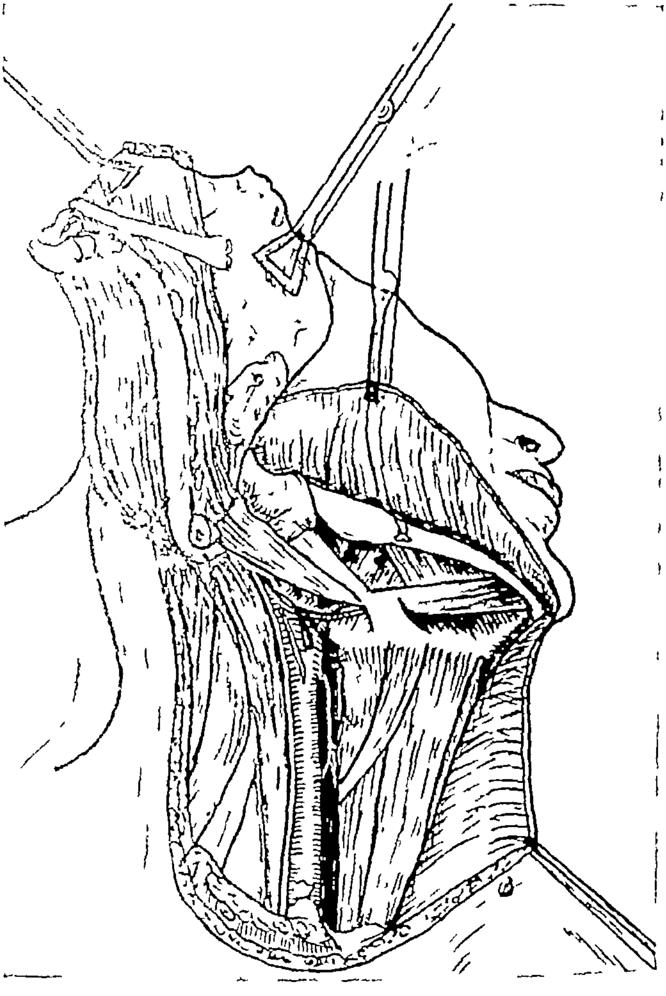

Fig 7 Showing extent of complete block dissection of the neck

5. The growth has invaded the thyroid cartilage or cricothyroid membrane.
6. In extrinsic carcinoma of the larynx.
7. Definite fixation of one or both vocal cords.
8. The growth is subglottic in origin.
9. In a recurrence following a laryngofissure operation.
10. Where the larynx has been subjected to unsuccessful irradiation in selected cases.

Also to be considered in these cases is the extent of the growth and its location. With a growth situated as above stated, a laryngectomy is the procedure of choice. These indications should be well understood; however, it is surprising that now and then we find a patient in whom conservative surgery has been used to the detriment of a lasting cure.

TREATMENT.

Complete removal of the primary growth is the utmost consideration in any operative procedure. The general physical condition of the patient must be such that he is a reasonably good operative risk.

The antibiotics unquestionably have been a great factor in postoperative results by keeping down infection.

The incision used is one best suited to the surgeon, providing it gives him adequate exposure, so as not to unnecessarily traumatize tissue. As I have already stated, the procedure must insure complete eradication of all diseased tissue, ranging from simple laryngectomy to complete laryngopharyngectomy; and if at all possible in the latter, to try to leave some normal pharyngeal mucosa for closure, instead of a large pharyngostome to be closed later by plastic.

PROGNOSIS.

The prognosis as to possible cures of these extensive lesions are in the following order: *1.* Cords; *2.* Epiglottis; *3.* Epiglottis and Tongue; *4.* Arytenoid; *5.* Ventricle; *6.* Subglottic; *7.* Pyriform Sinus; *8.* Aryepiglottic Fold.

SUMMARY.

1. The extrinsic carcinomas of the larynx have been broken down and placed in their anatomical sites.

2. The operative procedure for these extensive lesions must be laryngectomy, and at the time of operation sufficient contiguous tissue surrounding the carcinoma must also be removed to assure complete eradication of the disease.

3. If the lymph nodes are involved, they should be removed at the time of the original operation on the larynx.

4. In some cases it may be necessary to do a very extensive gland resection plus the larynx and enough of the pharyngeal tissue to insure adequate removal, which may result into a laryngopharyngectomy.

5. Results of these procedures are shown.

224 Delavan Avenue.

SYMPOSIUM: CARCINOMA OF THE LARYNX.

III — ROENTGEN THERAPY OF CARCINOMA OF THE ENDOLARYNX.*

J. A. DEL REGATO, M.D. (by invitation),
Colorado Springs, Colo.

In the past 30 years a slow but steady progress has taken place in radiotherapy of cancer. This progress has been due primarily to the development of rational techniques of treatment based on better knowledge of physics and radiobiology, to better dosimetry, to the technical perfection of equipment and to the advent of supervoltage Roentgen therapy. The progressive improvement of results in the treatment of carcinoma of the cervix and carcinoma of the lower lip, among others, have clearly shown this undeniable progress.

In these three decades there has been also remarkable improvement in the results of surgery of cancer. Surgical progress has been mostly due to better knowledge of anesthesia, to better pre- and postoperative care and to the advent of antibiotics, all of which have resulted in diminished operative mortality and enlargement of operability to include advanced lesions and aged patients (*e.g.*, cancer of the large bowel); and progress has been made also in the designing of new interventions (*e.g.*, pneumonectomy, esophagectomy), to be applied to previously inoperable tumors.

Progress in surgery as well as in radiotherapy of cancer has been in part due to wider understanding of the pathology of tumors, of their varied characters and life expectancy, of their invading and metastasizing habits; in other words, of their natural history. This common ground has brought about greater understanding of the possibilities and of the relative merits of both forms of treatment, resulting in considerable agreement among those interested in the nature of the disease first, and concerned secondarily with the character of their special skill; but, whereas the improvements in surgery have found immediate, enthusiastic, capable and vast application and consequent appreciation, the improvements in radiotherapy of cancer have not been rapidly assimilated or even recognized everywhere. The result has been a state of uncertainty and consequent overenthusiastic surgical excesses, advocated by the honestly uninformed or the youthfully inexperienced.

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*Read by invitation as part of a Symposium at the Fifty-fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 7, 1951

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One could easily save several times more lives by the simple expedient of introducing skilled radiotherapy to certain institutions than by the combined application of all other, considerably more expensive, available measures of detection and early diagnosis of cancer. In radiotherapy, as in any other medical specialty, a thorough adequate training is the shortest way to better performance; yet, most institutions in need of a therapeutic radiologist are obliged to choose a self-initiate for lack of sufficient numbers of well trained radiotherapists. As a result of a shortage of capable advocates, the responsibility for therapeutic radiology is disseminated in the departments of physics, dermatology, gynecology, hematology and even of plastic surgery, instead of being properly centralized under competent scientific control in the hands of those who understand the common principles of its varied application and its hazards; but, while all efforts are justifiably put to play in the endeavor to increase the results of surgery of cancer, departments of radiology are expected to carry on their duties without right to hospitalization of patients.

In otorhinolaryngology the rôle of radiotherapy has been definitely established in these three decades as the undisputed choice in the treatment of nasopharyngeal and tonsillar tumors, as well as of primary lymphosarcomas and undifferentiated carcinomas in any location. If the results of radiotherapy of these forms of cancer and of these regions have not always been remarkable, they are the only results obtainable and they require, at that, more than amateurish endeavors and skill; however, the treatment of cancer of the larynx has been the subject of uninterrupted honest disagreement for a long time. There is here also an area of possible agreement, but it is clouded by the inequality in experience with both forms of treatment, by the inequality in size and inequality of material available to different workers, by the inequality of segregation of patients by different authors and unequal use of certain words (*e.g.*, intrinsic) and by unequal reporting of results. In the presence of some of its most brilliant protagonists and advocates, I would not presume to analyze here the merits of surgery of cancer of the larynx. Allow me, therefore, to present only our views in respect to what we believe are its proper indications.

First, we should analyze the group of carcinomas of the *hypopharynx*. These tumors arise mostly in the pyriform sinus and may, though not always, invade the larynx secondarily; they are usually thrown in the large duffel bag of so-called "extrinsic" carcinomas of the larynx, yet they do not deserve to be called carcinoma of the larynx any more than a carcinoma of the stomach which directly invades the liver deserves to be called a hepatoma. We prefer to include also in this group the few borderline lesions which could be called laryngopharyngeal carcinomas, such as those arising in the free border of the epiglottis or arytenoepiglottic fold, which may extend both inside and outside the larynx. In this entire group of hypopharyngeal and pharyngolaryngeal tumors the accomplishments of radiotherapy have been rather poor. This has been partly at least due to the usual extensive development, frequent cartilage destruction and almost constant voluminous metastases which accompany these tumors. Any effort dedicated to improve these results either by means of radical interventions or any other means should be naturally welcomed.

Then we have the carcinomas arising within the limits of the larynx proper, or carcinomas of the *endolarynx;* these tumors are diagnosed relatively earlier, their extension is more limited and they metastasize less frequently than those arising outside the larynx. Both total laryngectomy and Roentgen therapy have been capable of curing a certain proportion of these tumors (see Table 1); but the results by either method have never been sufficiently satisfactory to justify its exclusive use. Radiotherapy has the advantage of being conservative, and when properly used it does not interfere nor handicap the possibilities of subsequent radical surgery. Total laryngectomy, while not offering much better results, condemns the patient to either artificial larynx or esophageal speech; this handicap is variously accepted or compensated by the patients and may be indeed a more serious mutilation than the loss of a limb, or of a lung. Without overplaying the drama of a mutilation produced by a curative surgical procedure, one must hesitate to impose it upon a patient when conservative procedures could be carried out successfully; lack of available radiotherapeutic skill has often given the necessary excuse.

TABLE 1.

RESULTS OF ROENTGEN THERAPY OF CARCINOMA OF THE ENDOLARYNX.

Author	Year	Cases	Lost to View or Dead of Intercurrent Diseases*	Well 5 Years or More	Absolute 5-Year Survival
Baclesse	1919 1940	215	?	48	—
Lenz	1931 1941	128†	15	23	—
Harris	1931 1942	80†	13	37	—
Cutler	1938 1942	107	7	37	—
Low-Beer	1936 1945	63	11	26	—
Total	1919 1945	593	46*	171	29%

*All these were considered as failures.
†Including some hypopharyngeal and laryngopharyngeal lesions.

A subdivision of carcinomas of the endolarynx into supraglottic, glottic and subglottic lesions may lead us to a fruitful understanding of choice indications of treatment. The *supraglottic* carcinomas, that is, those arising from the laryngeal wall of the epiglottis, the false cord and the laryngeal ventricle, are often relatively less differentiated tumors, they spread through the soft tissues anteriorly and laterally and frequently recur following surgical removal of the larynx and inadvertent incision through tumor. This group of tumors, in spite of their extension and cartilage destruction, may be successfully treated by means of radiations in a good number of instances, and for these reasons we feel that Roentgen therapy is their treatment of choice. Carcinomas of the *subglottic* region of the larynx are often differentiated lesions which rapidly occlude the air passage, necessitate a tracheotomy and often infiltrate the anterior wall of the esophagus. Roentgen therapy has been successful in the treatment of these subglottic lesions (Baclesse) ; but in general a laryngoesophagectomy offers greater chances of control, so that we feel that radical surgical removal is the treatment of choice of these carcinomas. Finally, we have the *glottic* lesions, or carcinomas of the vocal cord proper, which include the so-called "intrinsic" carcinomas of the larynx. Carcinomas of the vocal cord that are accompanied by edema or fixation of the larynx are infrequently controlled by Roentgen therapy, and we feel that they are best treated by total laryngectomy; on the contrary, small or exophytic lesions of the cords can be controlled with relative ease by Roentgen therapy (see Table 2), and in such cases we prefer this form of treatment in preference to total or partial surgical procedures. Lastly, we wish to reemphasize that when radiotherapy has been carried out adequately, recurrences may receive the additional full benefit of a total laryngectomy without hindrance.

We have said nothing of the technique of Roentgen therapy and unquestionably this is of paramount importance. We favor a protracted treatment spread over at least 10 weeks; treatments must be carried out under constant clinical control; fields should be adequate, square or round, but as small

TABLE 2.

ROENTGEN THERAPY OF EARLY CARCINOMA OF THE VOCAL CORDS.

Author	Year	Cases	Well 5 Years or More	Absolute 5-Year Survival
Baclesse	1919 1940	14*	10	—
Lenz	1931 1941	10†	8	—
Harris	1931 1942	8†	5	—
Cutler	1938 1942	15†	10	—
Low Beer	1941 1945	19†	15‡	—
Total	1919 1945	66	48	73%

*Limited to one cord.
†Suitable for "laryngofissure."
‡Four years' minimum control.

as possible; two lateral fields are preferable; well filtered radiations assure a more homogeneous irradiation; dosage will vary according to protraction, size of fields, etc., but usually several thousand Roentgens must be delivered to the tumor area; the dose administered is not an amount known to be the necessary one to destroy the tumor, but rather the maximum permissible dosage with safety to normal structures, under the circumstances of the treatment.

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SYMPOSIUM: CARCINOMA OF THE LARYNX.[†]

IV — CANCER OF THE LARYNX. FIVE-YEAR RESULTS.*

LEROY A SCHALL, M.D.,
Boston, Mass.

In 1947, we rendered a report[1] on a statistical study of patients with cancer of the larynx seen at the Massachusetts Eye and Ear Infirmary between the years of 1930 and 1945, inclusive. Although the group of patients seen between 1930 and 1940 had been followed five years and longer, we acknowledged that all of the group seen between 1941 and 1945, inclusive, had not, in 1947, been followed five years, and an evaluation of their treatment, therefore, was inconclusive.

This report will complete a study of the group of patients seen from 1941 to 1945, inclusive and followed five years or longer.

The number of patients with cancer of the larynx seen during this period and followed with accurate records was 228. In this series, the records show the type of treatment selected to have been:

External irradiation	132 cases
Laryngofissure	23 cases
Laryngectomy	60 cases
Cordal lesions—external irradiation	13 cases
Total	228 cases

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*Read as part of a Symposium at the Fifty-fifth Annual Meeting of the American Laryngological Rhinological and Otological Society, Inc., Atlantic City, N J., May 7, 1951

†From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary, Boston, Mass.

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The radiologic treatment of these patients with laryngeal cancer was carried out by the Department of Radiology of the Massachusetts General Hospital. Most of the patients received supervoltage irradiation to skin tolerance, receiving at least 6,000 r. or more tumor dosage.

In 1947, our results from external irradiation of laryngeal cancer as reported in our earlier communication were:

CANCER TREATED WITH EXTERNAL IRRADIATION.

Years	Cases	Dead	Five-Year Cure
1930-1935	24	18-75%	6-25%
1935-1940	75	58-77%	17-22.6%

The study of patients seen during 1941 through 1945 with an accurate follow-up on all cases treated by external irradiation reveals:

CANCER TREATED WITH EXTERNAL IRRADIATION

Years	Cases	Dead	Five-Year Cure
1941-1945	132	114-86%	18-14%

In addition to these cases of laryngeal cancer treated in our clinic, I have seen, in consultation, at least 10 patients treated at other clinics who had received from 10,000 to 12,000 r. of irradiation without control of the disease.

Laryngeal Cancer Treated by Laryngofissure:

In 1947, our results of laryngeal cancer treated by laryngofissure during the years 1930 to 1940 was only 58 per cent five-year cures in 31 cases. From an analysis of the records we came to the conclusion that in our clinic too many surgeons with but little cancer interest were not only selecting the patients for operation but were also doing the surgery. Since 1940, the cancer problem has been my responsibility. Although I have selected the patients and have either done or supervised the surgery, our five-year results have not improved.

CANCER OF THE LARYNX TREATED BY LARYNGOFISSURE.
1941-1945.

Five years or longer (no recurrence)	11
Dead from local recurrence	4
Dead from other causes—less than five years	2
Dead—distant metastasis—no local recurrence	2
Laryngectomy after laryngofissure—dead	2
Laryngectomy after laryngofissure—five-year cure	2
Total	23

Laryngeal Cancer Treated by Laryngectomy:

In the 1947 report, our results of total laryngectomy were:

LARYNGECTOMY

Years	Cases	Dead	Five-Year Cure
1930-1940	44	16-36%	28-64%

For the group of patients so treated between 1941 and 1945, inclusive, the results were:

Years	Cases	Dead	Five-Year Cure
1941-1945	60	27-45%	33-55%

Included in this series of laryngectomized patients there are nine cases in which irradiation failed to control the disease. These are not included in the table on the results of external irradiation.

LARYNGECTOMY AFTER X-RAY FAILURE.

Years	Cases	Dead	Five-Year Cure
1941-1945	9	9-44%	5-56%

Our results from laryngectomy in the 1941 through 1945 series are not so good as in the 1930 through 1940 series.

Perhaps this may be due to the fact that we have become increasingly bold in our surgical risks and are accepting cases that would have been considered inoperable in our earlier experience. Perhaps some of these cases would fall into a group that might be labeled "Salvage Surgery."

In the operation of total laryngectomy we have never performed the so-called narrow field of surgery. It has been our practice to do, what we like to call, a block removal of the larynx with its musculature. The prophylactic neck dissection as originally described by Sylvestre,[4] deserves serious consideration. We would like to restate our position concerning total laryngectomy from our 1947 report:

"The psychology of a surgeon dealing with cancer should be radical. The problem is how much can be removed and not how little. When a patient is faced with the loss of his larynx, he is not interested any longer in conservative surgery. The routes of extension of cancer of the larynx have been well demonstrated by Taylor and Nathanson,[2] and the importance of removing the pre-epiglottic space has been emphasized by Bisi.[3]

"In total laryngectomies, we have always used the 'T' incision or the 'U' incision of Gluck-Soerensen. The sternohyoid muscles and anterior belly of the omohyoids are removed with the hyoid bone or the body of the hyoid bone.

The pretracheal gland on the cricothyroid membrane is always examined microscopically for cancer. The pretracheal muscles are removed since they are rich in lymphatics and are in close relation to the pretracheal gland. We have found metastatic cancer in the sternohyoid muscle when, on gross inspection, it could not be detected. The thyrohyoid muscle is removed with the larynx.

"The carotid sheaths are always exposed and examined for involved glands and, should they be found, a radical neck dissection is done along with the laryngectomy.

"In cases of laryngeal cancer with unilateral cervical metastasis, Sylvestre[4] and Brunschwig[5] have demonstrated not only their operability but also an encouraging curability by radical neck dissection and laryngectomy."

In 1947, we reported on a small group of cases with cordal cancer which were ideal for laryngofissure but which were treated by external irradiation. We now have the five-year results of this group of cases:

CORDAL CANCER—IDEAL FOR SURGERY—
TREATED BY IRRADIATION.

Years	Cases	Dead	Five-Year Cure
1941-1945	13	1	12-92%

To complete and summarize our results in the treatment of cancer of the larynx for all patients seen between 1930 and 1945, on whom we have accurate follow-up and, therefore, followed for five years and longer, we report our results to be

CANCER OF THE LARYNX

	1930-1945.		
Treatment	Cases	Dead	Five-Year Cure
Irradiation	231	190	41-17.7%
Laryngofissure	54	22	32-59%
Laryngectomy	104	43	61-58%
Cord cancer—X-ray	13	1	12-92%
Total	402	256	146-30.7%

CONCLUSIONS.

1. Carcinoma of the larynx is still a serious disease and five-year results still depend upon early diagnosis and prompt treatment.
2. Statistics such as these serve only to record our personal experience.

3. Cancer is still unpredictable and cancer of the larynx still calls for a careful evaluation and individual consideration of each case. The type of treatment selected must be based on the pathology of the lesion, on the extent of the disease, on its duration and on the condition of the patient.

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SYMPOSIUM: CARCINOMA OF THE LARYNX.

V — SUMMARY OF CANCER OF THE LARYNX SYMPOSIUM.*

GORDON B. NEW, M.D.,
Rochester, Minn.

Dr. LeJeune has presented the treatment of early cancer of the larynx in a very thorough, concise manner, indicating the growths to be removed under suspension laryngoscopy and those requiring thyrotomy. I agree entirely with his indications and treatment. The results of the removal of these early selected cases by surgical diathermy using suspension laryngoscopy are excellent. There is good function without the dryness of the mucous membrane that I have seen at times following irradiation. These cases must be carefully selected by a laryngologist with a broad knowledge of laryngeal pathology and aided by an examination of fresh frozen sections of the tissue at the time of operation. Dr. LeJeune says that 6 to 7 per cent of his cases of cancer of the larynx are removed in this manner. From his description of the size of the lesions he has treated, I believe that even larger well localized growths can be safely removed in the same way. In a recent study 11 per cent of our cases were treated in this manner.

In mentioning some points in the technique of thyrotomy, he says that he has given up the use of the electric saw for dividing the thyroid cartilage in doing a thyrotomy as it at times produces a necrosis. This can be eliminated by having the first assistant direct a stream of cold water from a syringe

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over the cartilage during the process of dividing it. I prefer an electric saw to a hand saw, but use a cord-driven dental engine that works very satisfactorily.

The question as to making a tracheotomy, after the removal of a growth by means of thyrotomy, depends entirely upon just how much tissue is removed and the chance of a secondary edema developing with obstruction. Personally, I would rather make a few extra tracheotomies at the time of the operation than to have to do a secondary one after the patient is partially obstructed from edema. Postoperative emphysema of the neck, face and chest may be avoided by insertion of a split rubber drainage tube into the lower extremity of the wound down to the cartilage and leaving it for from 24 to 48 hours. All thyrotomy patients, as Dr. LeJeune has brought out, should be examined two months after operation for the removal of a possible granuloma that may be present, interferring with the perfect approximation of the vocal cords. In cases where both sides of the larynx have to be electrocoagulated at the time of the thyrotomy, one side may be malignant, and the opposite cord a thickened leucoplakia. Raw surfaces are produced on both vocal cords anteriorly that may cause an adhesion. This can usually be prevented by the insertion, at the time of the operation, of a soft rubber prosthesis covered by a finger cot, the same type as used in skin grafting the larynx, and suturing it in place through the skin with heavy sutures. This should be left in place two or three weeks and then removed from above. Holding the raw surfaces apart allows them to heal laterally. I know Dr. LeJeune will agree with me that in some cases an exploratory thyrotomy is indicated to determine whether the growth can be removed in a conservative way or whether a laryngectomy should be done.

I wonder whether Dr. LeJeune would tell us how he feels about the use of irradiation for epithelioma of the vocal cords.

Dr. Orton as usual has presented a very complete report on the indications for laryngectomy for extensive carcinoma of the larynx with his reasons for recommending that the prelaryngeal area, the hyoid bone and the pre-epiglottic space be removed *en masse* along with the larynx. He does a complete block dissection of the cervical glands if one is palpable.

To begin with, he accepts for surgical treatment 65 per cent of the cancer of the larynx patients he has seen, not including 76 patients who would not be operated upon. In my opinion this 70 to 75 per cent is about the right percentage of cancer of the larynx cases that can be benefited by surgical treatment.

He has divided the growths into eight locations as to the primary lesions, and his excellent results speak for themselves, showing what can be accomplished by untiring work of one interested in this field. He advises a cervical gland dissection when there is a palpable gland. Metastatic glandular involvement in carcinoma of the tongue usually involves the deep midcervical jugular gland, as in carcinoma of the larynx. The growth is usually more active. A group of cases in which a clinical diagnosis was made as to lymph node involvement in carcinoma of the tongue was recently reviewed. When there were no palpable nodes clinically, there were 22 per cent in which the microscopic examination showed involvement by malignancy at the time of operation. In those in which there were indeterminate nodes present, that is, where there was a question clinically as to whether or not they were malignant, 67.5 per cent were positive microscopically at the time of operation; so that we must assume that there is considerably more glandular involvement in these cases than is evident clinically.

I would think that Dr. Orton's end-result tables would be still more valuable if the cases in which cervical gland dissections were done were grouped together. These might be divided into two groups, those with and those without proven microscopic gland involvement. This would aid in showing what is accomplished by a prophylactic gland dissection in addition to the laryngectomy and also what end-result can be expected from a block dissection when a mid, deep cervical gland is involved. I would like to ask him if he has done gland dissections when there are *no* glands palpable; and whether there are any proven microscopically involved glands in those cases. Should all extensive cancer of the larynx have gland dissection regardless of whether any glands are found clinically? I would like to ask Dr. Orton if he uses radon seeds or irradiation in addition to surgery.

I did not receive Dr. del Regato's paper, but he wrote me a letter describing the indications for treatment of cancer of the larynx with irradiation; however, no mention was made of the end-results. Depending upon the location of the lesion, he divides the cases into four groups, which determines the method of treatment, but he does not mention any other factors such as the activity of the growths.

First, the small lesions of the vocal cords he prefers to treat with X-ray; "when they recur, a total laryngectomy is always possible without lessened chance of success." I am afraid I cannot agree with this statement. The secondary edema that occurs at times following irradiation makes it difficult to tell whether there is a recurrence present or whether the edema is the result of irradiation. At times an exploratory thyrotomy is the only way that one can be sure that there is a growth present and then it is found to be quite extensive. If a laryngectomy is necessary, the patient has lost the chance of a cure from a conservative operation which he would have had originally.

The second group of cases are "those of the glottis that have already fixed the larynx and which are accompanied with edema." He prefers to treat them by laryngectomy.

The third group of cases cases are "the supraglottic tumors which are less successfully treated by laryngectomy than by irradiation, so that the latter is the treatment of choice." I feel that the supraglottic carcinoma should be treated by teamwork of the laryngologist and the radiologist in order to give the patient the best chance to get well. These growths, of course, do not all require laryngectomies as he suggests. The growths of the epiglottis that are movable are best treated either by transhyoid pharyngotomy or, after a preliminary tracheotomy, by removal of the epiglottis with surgical diathermy under suspension laryngoscopy. Radon seeds are usually inserted at the time into the base and into the pre-epiglottic space and base of the tongue. A gland dissection is frequently done if the extent and type of the primary lesion warrants it. Fractional X-ray should follow this treatment so that everything possible is done. The patient has to wear a Rehfuss feeding tube and a tracheotomy tube for at least two months due to the reaction, but if the growth is all supraglottic and movable, the vocal cords are not injured so that a good voice results.

The fourth group, the subglottic growths, Dr. del Regato states should be treated by laryngectomy. In the letter no mention is made of end-results or length of time he has followed this particular method of selecting cases for treatment. Grading of cancers apparently is not taken into consideration. Would Dr. del Regato please comment on this?

I wish I could correctly evaluate Dr. Schall's results, either from the surgical or irradiation treatment of cancer of the larynx. In the text that I received, the indications for the selection of treatments were not given. In his conclusions, he states that cancer of the larynx is a serious disease and five-year results still depend upon early diagnosis and prompt treatment. I feel, and I am sure he does too, that the selection of the *type* of treatment for the individual case is very important factor in the end-results. In the 228 patients that he treated between 1941 and 1945, 145, or 63 per cent, were treated by irradiation. This includes 13 patients with tumors of the vocal cords. Only 83 patients, or 37 per cent, were treated surgically by laryngectomy or thyrotomy. This small percentage of cases selected for surgery should produce a higher than usual five-year cure, but it apparently has not. The 132 patients treated with external irradiation produced 14 per cent of five-year cures. This does not include the 13 patients with lesions of the cords who gave a 92 per cent five-year cure. The extent or activity of these growths is not stated. I am wondering if these 13 lesions on the vocal cords that were treated by irradiation and resulted in a 92 per cent cure might not be comparable to those early growths that we would treat under suspension laryngoscopy and surgical diathermy, or were they larger growths? I would like to ask Dr. Schall if he has changed his ideas as to the selection of treatment from the results that he has presented. Does he still feel that only a little over a third of the patients with cancer of the larynx should be treated surgically?

There is no one form of treatment that is superior to all others in all cases of cancer of the larynx, as has been brought out by these excellent presentations. On the contrary, the type of growth in question, its activity, extent, location and general condition of the patient should determine what type of treatment should be instituted. In general the low grade carcinoma should be treated surgically, and the extensive high grade growth treated by surgery and irradiation or irradiation alone. In general about 70 to 75 per cent of cancer of the larynx may, for the patients' best interests, be treated surgically. Irradiation has a very definite place as a therapeutic measure in the treatment of cancer of the larynx. I have seen patients with very active cancers, even with cervical metastatis, that could not be benefited by any type of surgery, who have remained well many years following irradiation. The cases selected for X-ray or radium should be just as carefully reviewed as those for surgery. Growths that recur after irradiation are very serious problems, as they are much more difficult to control and are much more extensive than is apparent. Prophylactic gland dissections have not been done on our patients, but block sections are done when a cervical gland is involved, and the primary lesion is surgical. Nineteen per cent of five-year cures have been obtained in patients with carcinoma of the larynx with cervical glands involved, proven microscopically.

Patients with carcinoma of the larynx usually have but one good chance to get well, so the laryngologist who determines the treatment must assume a serious responsibility. To tell a patient that conservative treatment may be tried and if this fails he still can have a radical operation usually does not result in the best interests of the patient. When the growth recurs, it is too late for any type of conservative operation that will leave the patient with a good voice, and the end-result of radical surgery is frequently not good. I am not referring to post-laryngectomy pharyngeal speech, which is an excellent substitute. In making the selection for the best treatment for the individual patient, we should all bear in mind the end-results that have been accomplished by the use of well established surgical methods with certain additions and modifications such as have been reported today. As I have stated before, irradiation has a very definite place in the treatment of cancer of the larynx. Further attempts to improve the surgical results are being made, such as prophylactic bilateral cervical gland dissection. Whether this will improve the five-year end-results, time will tell.

EXPERIENCES WITH PARENTERAL VITAMIN A THERAPY IN DEAFNESS AND TINNITUS.*†

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The anxiety of every otologist to find means to improve the hearing of his deafened patients makes him especially receptive to any new form of therapy that is alleged to be helpful. This anxiety, however, has usually been tempered with caution in view of past experiences with many forms of therapy that have been presented as successful, only to prove disappointing as time passed by.

Recently, favorable results have been reported in both deafness and tinnitus with the use of parenteral vitamin A. I am sure that many of you have wondered how substantial is this improvement and for how long it has or will last.

Lobel,[1] in May, 1949, presented a new injectable vitamin A preparation‡ which produced improvement in hearing and tinnitus in a considerable number of the approximately 300 patients on whom it was tried.

Subsequently, Anderson, Zoller and Alexander,[2] and Bau and Savitt,[3] using the treatment as outlined by Lobel, also reported high percentages of favorable results in patients with deafness and tinnitus.

This vitamin A preparation was made available to me§ in June, 1948, at which time I began my own studies. In reviewing my results and comparing them with those of the writers just mentioned, I am reminded of an incident that occurred a

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†From the Departments of Otolaryngology of the Stanford University Medical School and the Mt. Zion Hospital.

‡Anatola (Lobel), Parke, Davis & Co.

§Provided through courtesy of the manufacturer, Parke, Davis & Co.

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short while ago: a recent report on the results of X-ray therapy to the nasopharynx for epidermoid carcinoma revealed a higher cure rate than that of our own. A little perturbed, I asked our own X-ray therapist, Dr. Robert R. Newell, an outstanding man in the field, if a review of our own cases with the therapy given might not be indicated. He replied, "No, it's just the same old story—everybody can cure cancer but me." I feel, in this comparison, as did this distinguished gentleman—"everybody can cure deafness but me."

Lobel's studies were prompted by his belief in the relationship between hearing loss and nutrition, especially the vitamin A factor. He stated, "Mellanby[4] has shown that animals placed on an A-free diet develop degenerative changes in the auditory capsule, in Scarpa's and the spiral ganglion. Wolbach and Howe[5] observed that vitamin A deficiencies resulted in a metaplasia of the ectodermal leaf and a substitution of keratinized epithelium for stratified epithelium in the respiratory tract, ocular tract, paraocular glands, alimentary and genitourinary tracts. Failure in the reversal of these degenerative processes may be due to the inability of the body to utilize the vitamin A because of hepatic or various glandular dysfunctions, or the therapeutic time interval in different persons."

Lobel developed an injectable vitamin A preparation consisting of olive oil, terpins, and the "A" factor of a concentration of 50,000 units per cubic centimeter. He thought that the injectable preparation facilitated the absorption of vitamin A by the tissue in contradistinction to the uncertain absorption when vitamin A is administered in an oily vehicle only.

Lobel further stated: "Pharmacologically the terpins act as antispasmodics and analgesics and in this manner serve to initiate a large part of the amelioration of the ensuing tinnitus. The combination of the terpins, olive oil and 'A' factor is taken up by the blood stream. The vitamin A is absorbed and stored and the terpins are excreted in part by the upper respiratory tract and its adnexa. It appears, therefore, that by the manner of administration described, the reaction in

Scarpa's and the spiral ganglion, as observed by Mellanby, *is reversible** — the drug is made available to the middle and inner ear."

This statement, that the reaction in Scarpa's and the spiral ganglion is reversible by the administration of vitamin A, is *contradicted* by the experiments of Perlman.[6] Perlman, working on rabbits, confirmed Mellanby's experiments on young dogs fed on vitamin A deficient diets. Mellanby's dogs showed: *a.* nerve degeneration, more especially of the cochlear neurons; *b.* new bony growth in the modiolus; *c.* overgrowth of the internal periosteal layer of the capsule, that is, the bone in proximity to the brain; *d.* degenerative changes in the organ of Corti and sensory epithelium of the semicircular canals; *e.* occasionally a small amount of bony overgrowth was seen in the basal whorl of the scala tympani. Serous labyrinthitis as reported by Mellanby was not confirmed by Perlman.

After the animals in Perlman's experiments were depleted of vitamin A (see Fig. 1) they were fed with a vitamin A rich diet. This feeding, after depletion, did not restore the labyrinthine capsule to a normal state (see Fig. 2). Excess of periosteal bone on the internal surface remained but became more compact, and the large islands of vascular connective tissue were replaced by compact bone (see Fig. 3). The degenerative changes in the nerve fibres and ganglion cells were also not reversed. Secondary changes in the neural elements of the VIIIth nerve also showed no signs of reversal. Changes within the labyrinth appeared to be secondary to compression of the structures in the internal meatus by the new bone formation (see Fig. 4). Considerable compression of the cochlear nerve was found compatible with good auditory function as tested by the acoustic middle ear muscle reflex.

Much has been written concerning the possible relat
between vitamin deficiencies and deafness in hum
unequivocal, convincing evidence is still wanting.
incidence of acquired deafness in our own perso

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*The italics were inserted by this author.

spent prolonged periods in prison camps during World War II tends to discredit the influence of vitamins on hearing. A former resident in otolaryngology at Stanford University Medical School happened to have served with the Navy at an advanced base just after the hostilities with Japan ceased. He examined approximately 2,000 prisoners of war as they

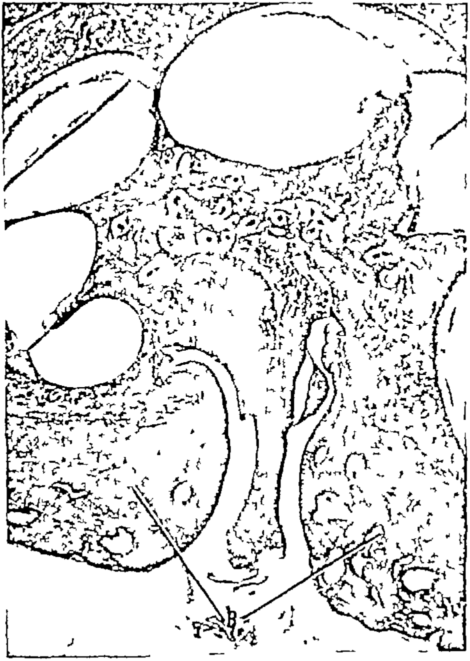

Fig 1 Rabbit, left ear—vitamin A depletion. New bone (B) thickens the internal aspects of the periosteal layer of the otic capsule with some lengthening and narrowing of the internal meatus. The new bone contains islands of vascular connective tissue which appear to have ossified in the reversal experiments. The line of demarcation between the old and the new periosteal bone is rather distinct (Courtesy of H. B. Perlman, M.D. Arch. of Otolaryngol., 50 22 July 1949)

were released from prison camps. Many of these had complaints and manifestations of vitamin deficiency diseases, but none of these included hearing changes. Examination of

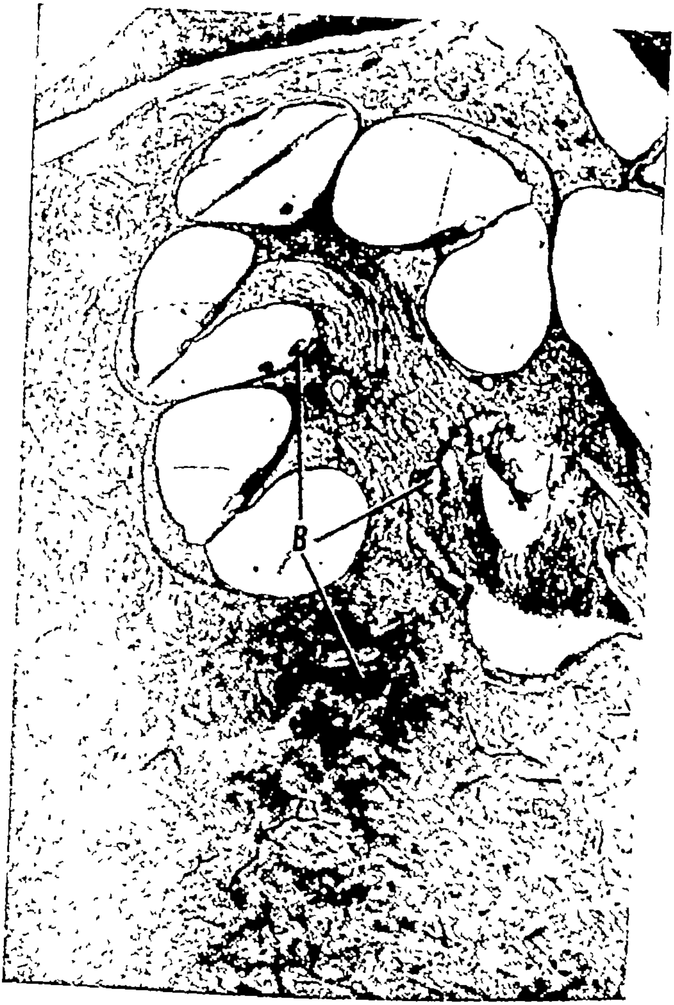

Fig. 2. Rabbit, right ear—vitamin A reversal. Widespread degeneration of cochlear nerve and ganglion, along with appearance of nodules of new bone (B) in the cribriform area and in the scala tympani. The periosteal and the enchondral bone are reorganized by dense bone, and there is hypertrophy of the posterior or intracranial layer. The nodules of new bone in the cribriform area probably encroach on the cochlear nerve and its vascular supply. (Courtesy of H. B. Perlman, M.D., Arch. of Otolaryngol., 50·28, July, 1949.)

hearing by gross tests (whispered voice) revealed no deafness in any of these individuals.[10]

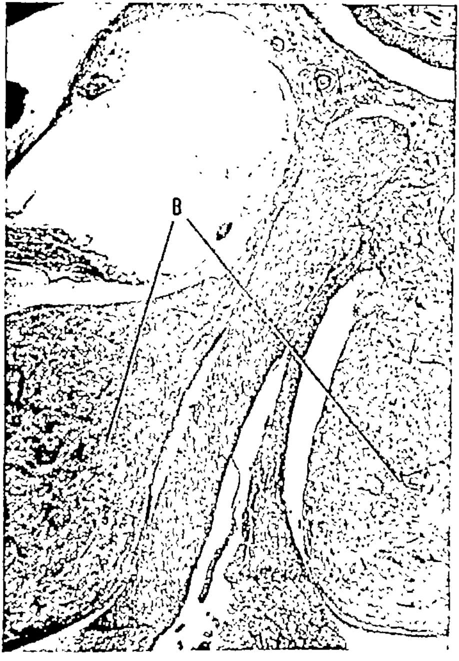

Fig. 3. Rabbit, right internal meatus—vitamin A reversal. The elongation of the meatus and the contained cochlear and vestibular nerve fibres is shown. The capsular bone is greatly thickened (B), but no connective tissue or fat marrow spaces are seen. There is no cement line of demarcation between the old bone and the newly proliferated bone of the capsule suggesting extensive reorganization of the capsule. (Courtesy of H. B. Perlman, M.D., Arch. of Otolaryngol., 50:26, July, 1949.)

The summary of Lobel's report is as follows:

1. Fifty thousand units of vitamin A were administered by the intramuscular route twice weekly for an initial period of

Sex: 21 females; 15 males.
Age: Nine to 72 years.

Types of Deafness:

1. Combined: 9
2. Perceptive: 10
3. Conductive: 17
34 were bilateral
2 were unilateral
  a. Other than otosclerosis: 6
  b. Otosclerosis 11 (of these, five had not had surgery, six had had fenestration operations by me or elsewhere, three successfully, three without success)

Tinnitus: 17.

RESULTS OF TREATMENT.

Five patients showed an improvement in hearing of more than 10 db for the conversational range (frequencies of 512, 1024 and 2048 cycles per second), but only three, or 8.3 per cent, of these had improvement not explainable by other factors. The improvement was in but one ear in two cases and both ears in the third.

One patient (No. 35) showed a slight diminution in hearing.

Of the 17 cases having tinnitus, four stated that their tinnitus was less. It was abolished in none. One patient had transient relief, two were temporarily worse during treatment and two developed temporary tinnitus that was not present before treatment.

Of the 44 patients who started treatment, 15 had reactions, eight of which were slight and seven were from moderate to severe. The slight reactions were redness and itching at the site of injection and were controlled with antihistamine drugs. The moderate to severe reactions included itching, redness, induration at the injection sites, nausea, dizziness and headache.

Evidence of hypervitaminosis A[7] was not noted. [This syndrome has been reported in infants and small children and

Name	Age	No. of Injections	Amt. of each Injection	Began	Ended	Ear	256		
							Before	After	Before
1 W B.	62	19	1 cc (50,000 u)	8/5/48	10/28/48	Rt.	25	20	30
						Left	23	20	35
2 E. B	58	22	1 cc	8/9 48	10 19/48	Rt.	15	10	10
						Left	30	20	25
3. Mrs. A. L. B.	70	20	1 cc	8/10/48	11 11/48	Rt.	45	35	30
						Left	25	40	35
4 D. C.	50	45	1 cc	8/8 48	12/23/48	Rt.	45	45	50
						Left	35	40	40
5. M C.	38	20	1 cc	7 10 48	9 9 48	Rt.	25	20	35
						Left	60	55	65
6. D T	37	20	1cc	6 8 48	8/20/48	Rt	40	35	50
						Left	55	45	60
7 R. F	45	36	1 cc	6 10 48	12/30 48	Rt.	30	30	25
						Left	65	35	60
8. M J	42	10	1 cc	10/11/48	11 12/48	Rt	30	25	30
						Left	50	40	45
9. D O.	20	15	1 cc	9/10 48	1/9 48	Rt	25	25	30
						Left	15	15	15
10. E. K.	47	20	1 cc	7 27/48	11 17 48	Rt	50	40	50
						Left	60	65	65
11. K. D	46	12	1 cc	8 11/48	1 4 48	Rt.	35	35	40
						Left	5	0	-5
12. J T	45	72	1 cc	9 24/48	1 13 49	Rt.	35	15	30
						Left	25	15	20
13. C. O	33	10	1 cc	11 1/48	12 10 48	Rt.	70	75	75
						Left	40	40	45
14 G. M	63	20	1 cc	1/19/49	4 7 49	Rt	25	20	30
						Left	25	10	25
15. A. W	40	42	1 cc	3/21 49	6 20 49	Rt	15	15	40
						Left	15	30	50
16. W W	70	14	1 cc	2/7 49	3 5 49	Rt	35	30	40
						Left	30	30	45
17 Mrs. A. B.	56	12	1 cc	3/22/49	6 4 49	Rt.	25	30	40
						Left	35	30	45
18. M C L.	45	19	1 cc	4/25/49	6 9 49	Rt	0	5	0
						Left	0	10	0
19 E. B.	38	19	2 cc (100,000 u)	6/9/49	8 5/49	Rt.	35	30	30
						Left	30	25	25
20. E. V	39	19	2 cc	7/25/49	9 14/49	Rt.	65	75	85
						Left	50	55	60
21 R. K.	34	20	2 cc	12/8/49	2/8/50	Rt	30	25	35
						Left	40	30	45
22. D L.	33	20	2 cc	2/20/50	4/24/50	Rt.	25	25	35
						Left	30	30	30
23 E. P	53	20	2 cc	4/13/50	6/19/50	Rt	65	65	65
						Left	15	20	25
24 Y M.	35	20	2 cc	1/16/50	3/27/50	Rt.	65	60	60
						Left	60	40	60
25. D W	36	28	2 cc			Rt.	30	30	35
						Left	35	40	45
26. E. L. N	60	19	2 cc	2/7/50	4/13/50	Rt.	5	5	5
						Left	45	45	60
27 H.	33	20	2 cc	2/7/50	4/27/50	Rt.	40	30	35
						Left	40	35	50
28. V S.	55	19	2 cc	4/13/50	6/16/50	Rt.	20	25	25
						Left	15	15	15
29. M. L.	42	16	2 cc	5/1/50	6/23/50	Rt.	10	10	10
						Left	15	15	10
30. Mrs. A. B.	16	19	2 cc	11/2/50	3/8/51	Rt.	30	35	30
						Left	30	20	35
31. S. R.	41	20	2 cc	12/12/50	2/20/51	Rt.	20	15	20
						Left	20	15	25
32. E. B.	53	20	2 cc	10/31/50	1/20/51	Rt.	65	65	70
						Left	50	40	40
33. R. C.	63	19	1 cc	3/11/49	5/5/49	Rt.	15	10	10
						Left	10	10	15

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ANDERSON, ZOLLER AND ALEXANDER.

	Right Ear Decibel Gain	Left Ear Decible Gain
Group A—7 Cases	16.6	0.0
	15.0	6.7
	6.6	15.0
	10.3	13.3
	3.0	12.0
	11.7	10.0
	11.6	5.0
Group B—3 Cases	5.0	10.0
	5.0	10.0
	10.0	0.0
Group C—5 Cases	Between 5 and 8.3	

series. The improvement in the B group is very slight, and the same may be said for a few cases in the A group.

3. *Bau* and *Savitt* have grouped their improved cases as follows:

BAU AND SAVITT.

Group B: 5-10 db gain—7 cases
Group C: 10-20 db gain—7 cases
Group D: More than 20 db gain—2 cases

In this series, too, the inclusion of the seven cases in Group B is open to considerable question.

4. *Baron* (see case reports).

Five patients out of 36 showed subjective and objective hearing improvement. Two of these (Cases 7 and 12) were excluded from the final calculations because of other factors related as follows:

Case 7: This is a man of 58 with Paget's disease of at least 10 years' duration. There was an improvement of 28.3 db in the left ear after 36 injections; however, he had shown an equal improvement in the right ear before injections were started. This man had had episodes of good and bad hearing. It is in such a case as this that the interpretation of the effect of any medication must be guarded. The comments of this patient following the injections were interesting. At the end of the third injection he stated that he "gets a sore throat an hour following the injection, which lasts approximately one day." The first eight injections were given in the buttocks, the ninth was given in the arm. He stated that "the shots seem more effective when given in the arm." These remarks are further evidence of tremendous psychic factors which may be involved in any form of therapy, especially in that by injection.

Case 12. This 65-year-old lady showed a 10.6 db improvement in her right ear after injections, but she had already gained more than 10 db in her left ear by local therapy, such as reduction of her nasal congestion by local treatment, Eustachian catheterization and inflations and administration of the antihistamine drugs.

Case 31 was included in those with improved hearing with reservation since the only improvement shown was a 35 db gain in but one frequency (2048) in one ear. Such a gain is far from conclusive.

Case 36 (see Fig. 5) was the only one in the whole series of 36 that showed a dramatic change in her audiogram. This was a very tense, nervous woman of 53 who had a slight head tremor. There was some bogginess and pallor of her nasal turbinates and her nasal smears showed two plus eosinophiles. She was given an antihistamine drug and on several occasions her Eustachian tubes were catheterized and inflated. This resulted in no improvement in her audiogram. She was then started on vitamin A injections, 50,000 units each three times a week for two weeks and then 100,000 units twice a week. After the twentieth injection her audiogram showed a 25 db improvement in the right ear and a 23 db in the left. The patient developed a cold at about the time she finished her thirtieth injection, and an audiogram taken then showed that the curve had receded practically to the original level. Patient was given an additional six injections of 2 cc. each and then went on her vacation. Another audiogram taken two months later, or approximately six months after injections had started, showed a further hearing improvement of an addition 8 to 10 db. The total improvement since starting the injections was 33.4 db in the right ear and 30.6 in the left. Because of the spectacular improvement in the audiogram, it was decided to have the patient checked at the San Francisco Hearing Center. It is noteworthy that the tester there remarked, "The patient seemed nervous and ill at ease throughout the test." The audiogram done there showed a considerably lower level for both ears than ours done six days previously (see Fig. 6A). The reading showed an improvement of only 6.7 db for the right ear and 6.6 for the left. We again rechecked this patient four days later at my office and found that the audiogram was almost the same as the one we had done two weeks previously. I may state here that from time to time we have checked our audiograms with those of the Hearing Center and in most instances the two have run parallel.

Another thing of interest happened to the patient toward the end of her treatment. She lost her head tremor and became more relaxed. She said that she "felt very much better."

It is interesting speculation as to the *modus operandi* of the vitamin A in this patient's improvement. It seems apparent that it was not through a selective action on the cochlea. It seems more likely that the preparation, through its effect on the patient's well being, removed the influence of the psyche upon the auditory mechanism. This patient became accustomed to going in and out of our office and was always relaxed and at ease when audiograms were done, whereas when she went to the Hearing Center she was anxious, nervous and ill at ease. This difference in mental state was able to produce an approximate 25 db difference in the audiograms on the two occasions.

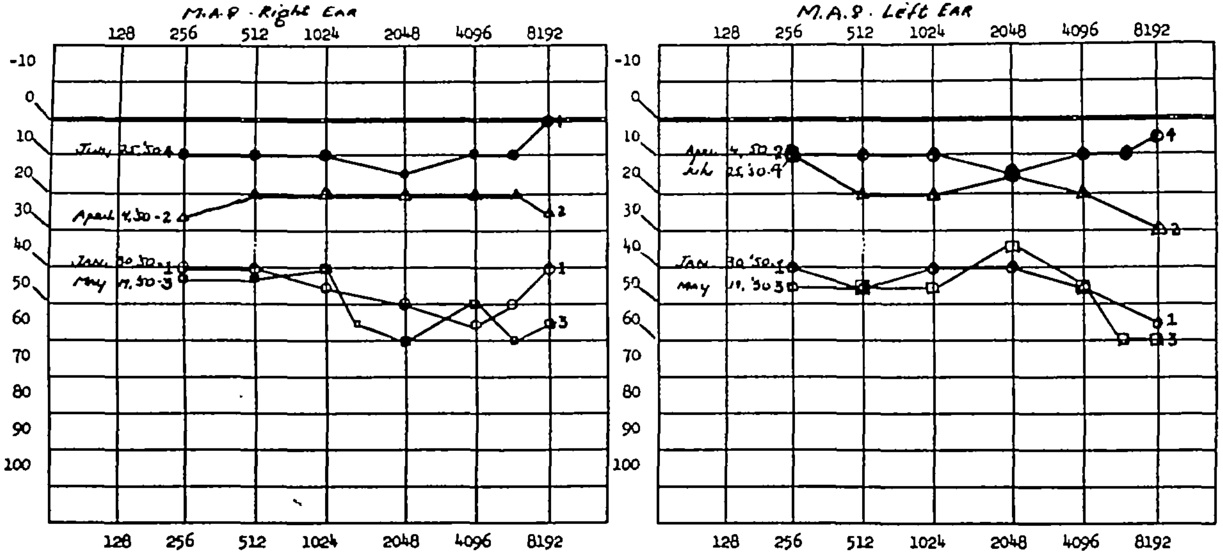

Fig. 5. Audiograms of Case 36. (1) Jan. 30, 1950. (2) April 4, 1950. (3) May 19, 1950. (4) July 25, 1950.

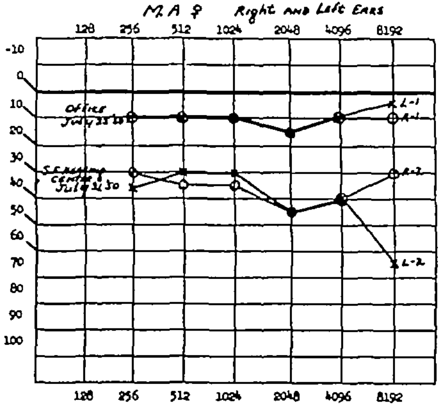

Fig 6A.

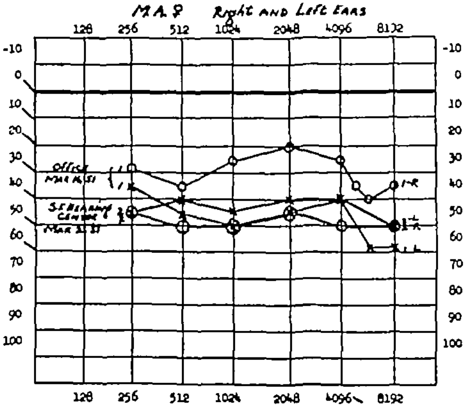

Fig 6B.

Fig 6A. Case 36 Difference in audiograms done six days apart by different testers with different audiometers

Fig 6B. Case 36 Eight months later Difference in audiograms done five days apart by different testers with different audiometers.

At this point I should like to present the audiograms (see Fig. 7) of a patient treated by another method, that of the aminoacid and vitamin treatment of Hirschfeld, Jacobsen and Jellinek.[8] For a short period, as a matter of interest, I was alternating patients between this substance and parenteral vitamin A.

The audiograms just mentioned are those of a retired naval officer who complained of nausea, vomiting, occasional dizziness and deafness in his right ear of about three months' duration. After a thorough examination was done to rule out any significant pathology, the patient was started on aminoacids and vitamins by mouth and by intramuscular injections. After about three weeks of this, he stated that he thought his hearing was worse. Another audiogram was done at this time

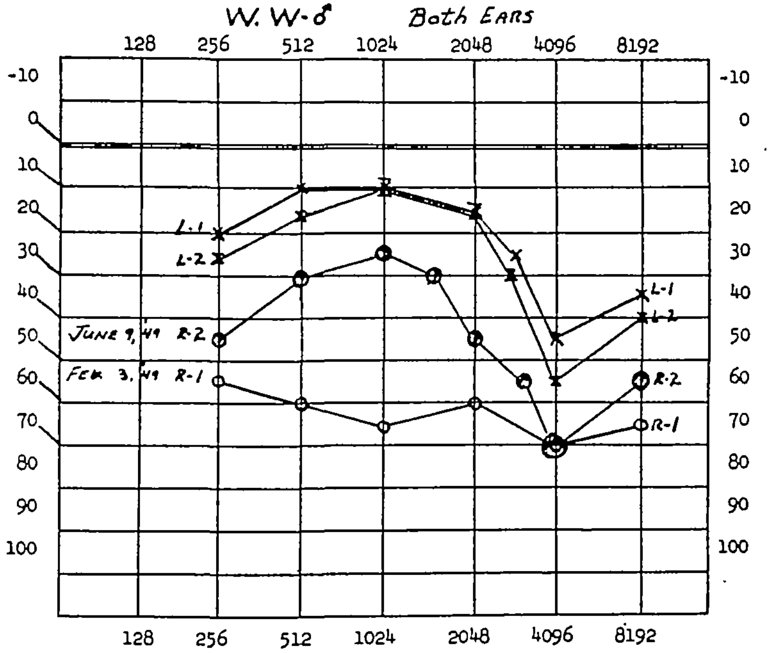

Fig. 7. Audiogram showing improvement in hearing in the right ear of a patient treated by another method (aminoacid and vitamin injections.)

and showed a 35 db *loss* in his left ear and a 20 db gain in his right ear. Recheck examination revealed a low-grade suppurative maxillary sinusitis for which local treatment was given In addition, the Eustachian tubes were catheterized and inflated at repeated intervals, and the administration of the vitamins was continued. At the end of three months the audiogram showed that the hearing in the left ear had returned to its previous level while that for the right ear had improved 27 5 db.

By chance of the "draw" this man was given injections of aminoacids and vitamins rather than parenteral vitamin A. The results from both probably would have been the same and an enthusiast for either medicament might have attributed the improvement in hearing to the drug. Actually, this improvement was due, likely, to the reduction of the inflammation in the nose and Eustachian tubes by the treatment of the sinusitis.

COMMENT.

It has not been my intention to discredit the conscientious reports of others who have had such glowing, favorable results in both hearing and tinnitus from the use of parenteral vitamin A. I did feel it a duty, however, to present the other side of the picture as shown by my experiences with the same therapy. Although the results of the various writers are three to one against me, it is difficult to understand why the patients in the east responded better than did those in the middle west, why those in the middle west did better than those in the south, or why the people in all sections responded better than those in the San Francisco area.

I doubt that vitamin A, presumably made more available to the body tissues by injection, produced improvement in any patient by a specific action on any portion of the auditory mechanism. It is conceivable that there may have been instances of improvement secondary to that of the upper respiratory mucosa in vitamin A deficient individuals in whom administration of vitamin A may have reversed epithelial changes.

A probable explanation for improvement in some patients was their increased resistance to fatigue, which is known to play an important part in loss of auditory acuity.[9] Psychogenic factors have already been referred to and undoubtedly played a dominant rôle in many of these treated patients.

CONCLUSION.

The administration of vitamin A by intramuscular injection in a series of 36 patients having all forms of deafness and tinnitus coincided with improvement in deafness and tinnitus in very few of these cases. This injectable vitamin A has not, in my hands, shown a superiority over other varied forms of therapy presently available.

SUMMARY.

1. Results of the study of 36 patients with varying forms of deafness and tinnitus treated with intramuscular injections of vitamin A have been reported. There were but three patients with improved hearing and four with lessened tinnitus. (Two additional patients showed an improvement in hearing of more than 10 db but were not included because there were other factors which definitely contributed to this improvement. Table I shows other cases with an improvement of around 5 db which were not included because they fell within the range of normal error or variation.)

2. The experiences of others (three groups) have been reviewed. All reported results better than those in the series just presented.

3. The experimental work of Perlman revealed that the damage done to the internal ear by vitamin A depletion was *not* reversed by vitamin A excess.

4. Vitamin A administered intramuscularly for the treatment of deafness and tinnitus in this report showed no superiority over other forms of therapy presently available.

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10 As told to Dr R. C McNaught.

BULBAR POLIOMYELITIS:
A RESPIRATORY PROBLEM.*†

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Further experience with bulbar poliomyelitis has confirmed our previous conviction that secretional obstruction, anoxia and carbon dioxide accumulation are much more important in this disease than the direct action on the vital centers of the virus itself;[1] and we believe more firmly than ever that proper treatment based on that idea can prevent many more deaths in this disease. Because some men speaking with authority and basing their conclusions in part on laboratory rather than clinical data are still skeptical, it seems worthwhile again to present the reasons for that concept, the treatment dictated by it and the results of that treatment. Men with experience with respiratory obstruction seem easily to see the logic of these ideas; some others cursorily dismiss them with little to offer in their place.

Bulbar poliomyelitis has many factors in common with other respiratory obstructions. Some of these are: the identity of many symptoms with those of anoxia and carbon dioxide excess; the rapid worsening at a critical level after a period of relatively moderate difficulty; the rapid amelioration of such symptoms when the airway is cleared before the stage of irreversibility is reached; the large percentage of recoveries based on treatment directed to the relief of obstruction.

Treatment based on this belief resulted in no mortality in 12 successive patients with pharyngeal paralysis treated early

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†From the Northwestern University Medical School and the Evanston Hospital.

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under good control at the Evanston Hospital in 1947 and 1948 (see Table 1). At the same time in Illinois, outside of Chicago, the mortality was 38.8 per cent in 105 bulbar cases. Some more recent cases were unfavorable because they were transported greater distances and were seen later, but our four-year mortality, 1947-1951, was 12.8 per cent in 63 bulbar cases, while at the same time the mortality in Illinois was 38.3 per cent in 722 bulbar cases. Our series is small statistically, but Prof. H. T. Davis estimates that the probability of such results occurring by chance was not more than six in 10,000. When the same general principles have been followed as by Bower[3] and his group at Los Angeles and by Priest, Boies and Goltz[4] the results were generally considerably better than the national average.

TABLE 1.
MORTALITY IN BULBAR POLIO.

Locality	Years	Total Polio	Bulbar and Bulbar Spinal	Bulbar and Bulbar Spinal Deaths	Bulbar and Bulbar Spinal Rate
Evanston Hospital	1947-48	127	15*	0	0
Evanston Hospital	1947-50, incl.	314	63†	10	12.8%
Chicago Residents	1948	312	37‡	10	27
Illinois, outside Chicago	1947-48	1,243	105	39	38.8%
Illinois, outside Chicago	1947-50, incl.	5,018	722	277	38.3%

*Three of 15 had no pharyngeal paralysis.
†Twenty-five had tracheotomies.
‡Of 12 additional bulbar cases originating outside Chicago, nine died.

For the understanding of bulbar poliomyelitis as a respiratory problem it is necessary to consider: 1. the effect of anoxia and carbon dioxide accumulation and 2. other effects secondary to respiratory obstruction.

Anoxia is used in its accepted sense to include carbon dioxide excess. How quickly it may produce disaster is still not widely appreciated. We have shown elsewhere[5] that 60 seconds of anoxia may produce morphologic changes in the a degree often requiring use of the respirator, but this is usually partly or wholly relieved after anoxia is relieved. Bower[3] states that most of the so-called central arrythmias likewise clear up after acidosis is remedied.

Material of the pathologist comes from patients who died with anoxia — if not because of it. Others who certainly faced that outcome with symptoms going from lethargy to coma, and early vasomotor and respiratory collapse have been decisively saved by treatment from imminent death and thus furnish no pathological refutation. Nothing in *a priori* reasoning seems to match the evidence given by the abrupt change in clinical course produced by clearing the airway. Six of the 12 consecutive patients with bulbar polio who did not die surely would have done so, and quickly, without such intervention.

To hold that the severe effects in bulbar poliomyelitis are due only or even chiefly to overwhelming virus infection of vital centers leads to defeatism and a negative approach to treatment that offers little or no help in the disease. Even if some of the views herein expressed prove later not to be wholly true they offer at present about the only working hypothesis of promise. They will hardly be dismissed without trial as oversimplification by a group who know the problem of respiratory obstruction and the satisfying methods of its relief. In any event, obstruction does exist in these cases from secretion and spasm or paralysis of larynx and pharynx and must be corrected if these desperately ill patients are to survive.

Three clinical types of respiratory difficulty are generally recognized in acute poliomyelitis:

1. Spinal paralysis affecting intercostals and diaphragm. These patients fall easily into rhythm of the respirator and do well in it, but may be permanently disabled.
2. Bulbar paralysis.
3. Bulbospinal.

Disturbances in rhythm, depth and coordination have been ascribed to infection of specific respiratory nuclei in the medulla. Such effect can occur but we believe it rare. We believe such disturbances are most often a result of conflict between ventilatory demand and protective mechanisms to prevent self-drowning, plus the effect of anoxia and carbon dioxide on the medulla. We believe this because we have seen the irregularity abolished by pentothal sodium (with serious results) and because it usually disappears on clearing the airway if not done in a terminal stage.

After tracheotomy and the respirator, our first patient seen with bulbar poliomyelitis went from coma, extreme cyanosis, imperceptible pulse and arrest of respiration for three minutes to rationality, normal color and pulse in 45 minutes. We have had so many patients with almost as marked recovery in as a short time that it is hard to believe that the condition could have been due to anything but asphyxia. Certainly such a rapid course does not seem compatible with any known type of infection.

In bulbar polio conditions are almost ideal for the production of anoxia. Due to inability to swallow, 1,500 to 2,000 ml. of saliva per day plus inflammatory secretion and food or vomitus may accumulate in or over the airway. We have recovered by aspiration over 2,200 ml. in 24 hours. This fluid mechanically impedes air flow, produces spasm of the larynx and tends to be drawn in with inspiration, especially in those instances where the paralysis destroys the protective function of the larynx. Flooding may occur because in the ordinary supine position the declivity of the tracheobronchial tree is from 17 to 20 degrees so that secretion flows with gravity or is easily drawn into the lung bed.

It has been common in our patients to find considerable accumulation of secretion in the pharynx, trachea and bronchi along with symptoms of anoxia. It has also been the rule that unless anoxic symptoms have persisted too long, they are relieved relatively soon after clearing the airway. The symptoms of medullary depression have usually followed milder symptoms of anoxia and also are usually relieved by clearing the airway, suggesting as Bower[3] states, that this is due to acidosis or anoxia.

We believe, then, that the primary difficulty in bulbar poliomyelitis is difficulty with the swallowing mechanism with accumulation of secretion and food and vomitus in or over the airway. Cough and other expulsive mechanisms are ineffective and unless fluid is kept removed by treatment it will lead to anoxia by preventing ingress of air, by producing laryngeal spasm, by blocking bronchioles, and by flooding and secondary damage to the respiratory area of the lungs.

In 1929, J. I. Durand[13] observed that in his opinion "many polio deaths are due to drowning as many men must have observed," and he made the very vital recommendation for postural drainage. The analogy is good, since except for speed the two processes are very similar. As Motley[14] points out, laryngeal spasm is often a very important factor in the suffocation of drowning. It has seemed that spasm may be important in bulbar poliomyelitis and that it is more likely to be a factor than obstructive median paralysis of the vocal cords. We are not sure we have ever seen this in polio. Also, as Furstenberg[15] has pointed out, nuclear or infranuclear paralysis of the cords is flaccid and not spastic. Sjoberg[16] postulates that a combination of spasm and paralysis of tongue and pharyngeal muscles with the head extended blocks the airway, although we have not been able to note that in obstructed cases.

The comparison to drowning is useful since the time element may be as short and the moment for action may as quickly pass. Seconds count in any form of asphyxia. Ignorance or indecision and inertia may hold to stupid inaction a man who would without hesitation leap into a raging torrent to save a child in hardly graver danger.

Treatment of these cases may be relatively simple or hopelessly involved. If undertaken early, decisively and with a visualization of the problem, it usually offers little difficulty although it requires meticulous and unremitting care. If delayed by lack of opportunity or indecision, it may require the utmost in experience, skill, team work and equipment. The simpler class of cases should be cared for by any well trained otolaryngologist with the aid of a good medical man or pediatrician if he gets an early chance, although decisions are made too frequently without consulting him by men who have not the experience or imagination to understand the problem.

Postural drainage and continuous suction, best obtainable by a Venturi type pump, are in our opinion the most important steps in treatment. When the patient first gives evidence of pharyngeal paralysis by muffled voice and inability to swallow secretions, even though he can apparently cough or push them out of his mouth, he should be placed with the feet elevated and the neck extended so fluid may accumulate as in a sump, at the nasopharynx or hypopharynx where it may be picked up as quickly as it covers the opening of a perforated rubber tube connected to continuous water suction. Remembering the few minutes of anoxia necessary to produce irreversible damage, it must be obvious that dependence cannot be placed on intermittently applied mechanical suction. We know of no mechanical apparatus which will bear constant use except one attached to a vacuum tank. The device pictured (see Fig. 1) is easily attached, effective in the pressure desired, almost foolproof and does not get out of order. Some plumbing codes interdict its use, yet if it is connected to a half liter collecting bottle filled with antiseptic solution, contamination of the general water system seems impossible.

Thirty years of experience with postural drainage in respiratory obstruction, especially in acute infectious tracheobronchitis, has persuaded us that it is of the utmost value and without danger if used early. Lateral X-ray with radio-opaque oil outlining the tracheobronchial tree[1] indicates that elevation of the foot of the bed in the supine position must be at least 17 to 20 degrees to prevent flow of secretion into dependent lung bed. We have advocated 35 degree elevation but now believe that extreme, and seldom obtainable. Twenty-three to 5 degrees is usually satisfactory. If the patient will lie prone with the head to the side, as usually only infants will do, drainage is effective and normal physiology is not disturbed. Adults may comfortably maintain such position if sustained by special sponge rubber pads.[16]

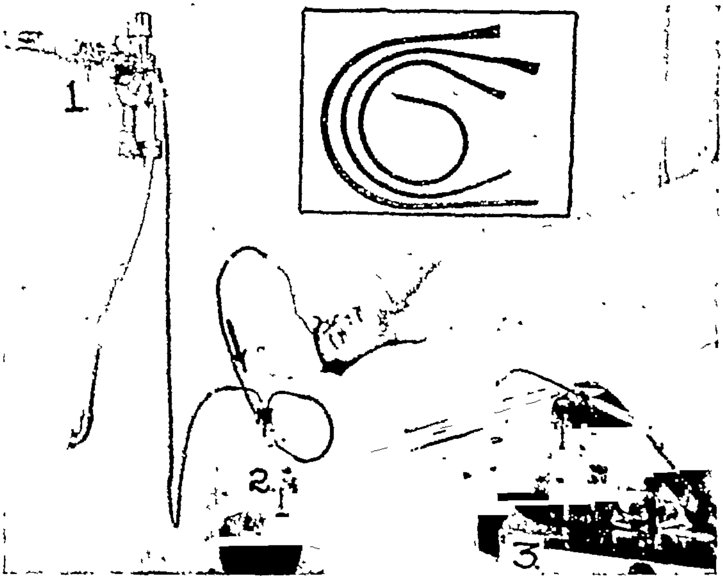

Fig. 1. (1) Is water suction (supplied by Surgical Mechanical Research, 1905 Beverly Boulevard, Los Angeles 4, Calif.) fixed to the wall, connected by fairly stiff tube to collecting bottle (2), this to catheter through patient's nose to the pharynx. (3) Is a standby mechanical suction. Beveled and whistle tip catheters are shown in inset.

Bower and his group[3] have raised a question as to the safety of the Trendelenberg position in seriously ill patients. At 15 degrees' elevation for 15 minutes the average drop of tidal air was 20 per cent. This, however, left an average tidal air of over 300 ml. on which a patient at rest may be carried, especially if adequate oxygen is given. This objection does not seem to outweigh the great benefit of postural drainage, which helps insure that the lower airway is not flooded, the alveolar area is accessible and the absorption of oxygen and excretion of carbon dioxide well distributed throughout functional lung. It is doubtless true that in patients with marked vasomotor disturbance caution must be used against extreme position.

Fluids and nutrition are maintained parenterally while there is *any danger of flooding the airway. It is especially* important that electrolite balance is strictly maintained. Bower and his group have pointed out that potassium deficiency is likely to occur in polio with loss of sodium balance and also with weakness of the cardiac and respiratory muscles. This may be very important, as shown recently by Darrow and Pratt.[8] This may arise from cell destruction, from acidosis, and probably through saliva lost. Potassium can be given intravenously with safety only with careful blood studies, which are hard to secure; however, it can be given subcutaneously with relative safety in Darrow's solution. The proper concentration will be picked up through the gastrointestinal tract if given by mouth. For this reason early feeding through a tube to the stomach is advocated by the Los Angeles group. This also permits aspiration in the not infrequent gastric hemorrhage. On account of the danger from vomiting, we still choose to use only parenteral administration of fluids, glucose, electrolites, vitamin C and B complex *and protein hydrolysate until patients can swallow*, although if tracheotomy has been done, tube feeding is not especially dangerous, since regurgitated fluid is then less likely to be aspirated.

Preventon of avitaminosis and hyproteinemia are essential for recovery from tissue damage, to prevent pressure sores and to promote healing of tracheotomy or other incisions. Proper hydration is especially important to keep secretion thin and easily removable. Excess of fluid may promote cerebral or pulmonary edema, but allowance must be made for the 1,500 ml. or more lost by saliva, regurgitation and other secretions.

Tracheotomy was first suggested for this condition by Wilson,[19] in 1932, but only in recent years has it come to be widely accepted. The visualization of conditions making it necessary, its precise indications, its harmlessness and frequently lifesaving necessity are well appreciated. Probably its most important benefit is the short circuiting of pharyngeal secretion and the prevention of aspiration as shown in Fig. 2. The suctioning of the secretion already aspirated through the

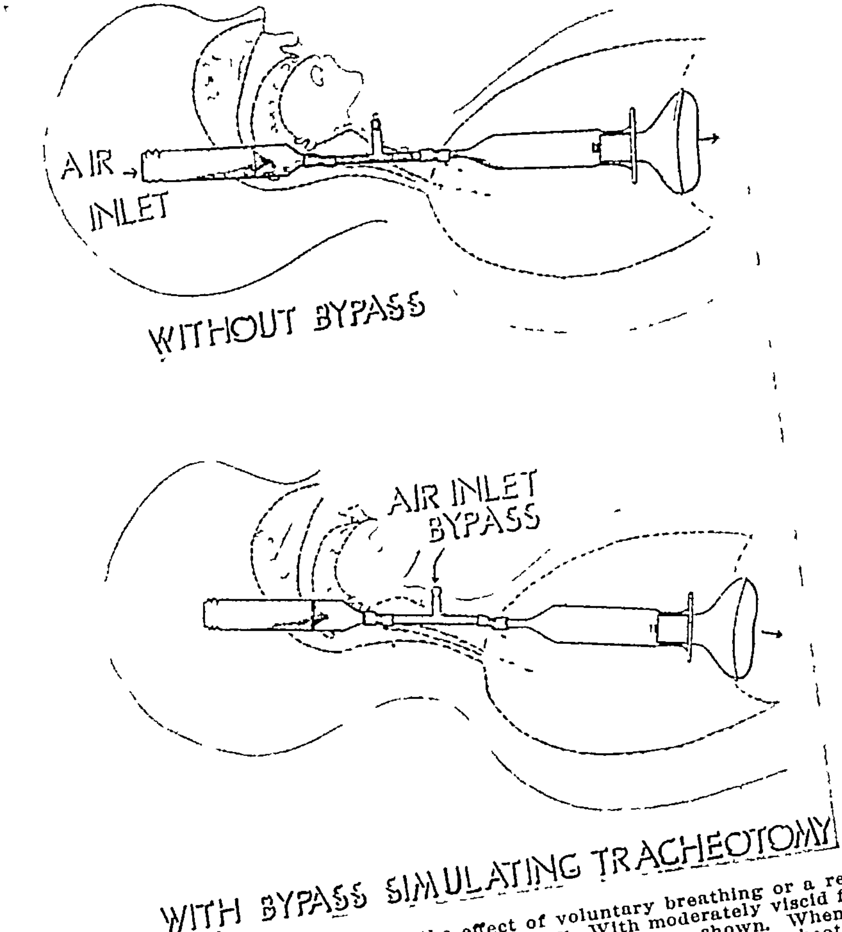

Fig. 2 A model to simulate the effect of voluntary breathing or a respirator when there is fluid in the upper airway With moderately viscid fluid, intravenous tube, T tube and aseptosyringe it acts as shown. When the by-pass is open no fluid is drawn in—illustrating the effect of tracheotomy. (Reproduced from Archives of Otolaryngology, 46:125, Aug., 1947.)

tracheotomy wound, and the provision of a clear airway are especially valuable. Since widespread use was advocated in 1943[1] with an attempt to rationalize this procedure statistics of large numbers of operations by Priest, Boies and Goltz,[4] by Bower,[3] and others have shown its great value and freedom from danger. Our indications as previously stated are:

1. Progressive anoxia with secretion in the airway.
2. Coma or other sign of anoxia without relief in a few minutes.
3. Marked restlessness or stupor in a patient already in a respirator.
4. Difficulty with secretion in a respirator patient.
5. Bilateral spasm (or paralysis) of the larynx.
6. Rapidly progressive bulbar symptoms, usually due to anoxia and carbon dioxide.
7. Untrained attendants, inadequate equipment or poor cooperation with any doubt that the airway will be kept free of secretions.

Reasons for these indications were discussed in earlier papers.

We are not quite ready to recommend routine prophylactic tracheotomy if the clinician is prepared to intervene at any moment and realizes how rapidly such patients can fail. It is much better to do tracheotomy unnecessarily in many cases rather than too late in a single one, and too early rather than after irreversible damage. We have seen no important complication from the operation, nor any case in which it contributed to an unfavorable outcome. It is not to be withheld as it is too often for the last forlorn hope. It should be done more often with a physician's first experience with the disease as much greater skill, training and equipment are required to carry on without it. It should be done high, through the second interspace and third tracheal wound to permit use of the respirator.

Postoperative care of tracheotomies is especially important since in these patients cough is impaired or absent, especially if they are in a respirator. Great gentleness to avoid trauma is required. While continuous water suction is continued through the nose, intermittent suction is used through the tracheotomy tube, removing the inner cannula if necessary. A soft whistle tip catheter is gently used or a soft rubber catheter obliquely cut and smoothed in a gas flame with a small counter opening near the end to break excessive grasp of mucosa, while permitting removal of secretion or plugs. Dew point humidification, use of wetting agents, gentle irrigation with warm 3 per cent soda bicarbonate solution followed by normal saline, both removed by suction in postural drainage will usually prevent atelectasis and prevent the need of bronchoscopy. Forceps technique is employed for asepsis, antibiotics are used but not long enough to permit the overgrowth of monilia and other fungi.

Treatment at the Evanston Hospital, which gave no mortality in 15 consecutive bulbar polio cases, and an allover mortality of 12.8 per cent for four years, is outlined:

1. Postural drainage with continuous water suction instituted at beginning of swallowing difficulty.

2. Parenteral fluids, electrolites, vitamin C and glucose followed after the third day with intravenous B complex and protein hydrolysate or amino acids.

3. Oxygen administration, but as an adjunct to clearing the airway and eliminating carbon dioxide.

4. Tracheotomy when signs of anoxia persist or develop in spite of above.

5. The respirator when respiration is depressed from anoxia, exhaustion or any other cause.

6. Antibiotics against infection (but not to the point of overgrowth of fungi, which may explain some troublesome exudates).

Such treatment could easily be adopted by any member of this group and should be usually effective if opportunity were given early.

Certainly even without special experience with the disease these measures are to be preferred to special apparatus which perhaps places wrong emphasis and delays proper measures. Some of these need special discussion as some are very valuable, especially in serious cases

Oxygen is of proven value in respiratory obstruction It should, however, be emphasized that much more important are the measures taken to provide for the elimination of carbon dioxide A clear airway is the prime need and no special device to provide oxygen should divert attention from this.

The oximeter has been proposed to determine oxygen need It needs to be set against a normal base, is not a thoroughly reliable apparatus, gives no direct indication of carbon dioxide excess and in our experience is only of value in determining when a chronic respirator patient may leave the respirator.

The electrophrenic respirator has some value in emergencies but does not have the applicability or dependability of the tank respirator It is not well tolerated for long periods and does not seem to warrant the emphasis given it. Bower[20] tried the method and discarded it as impractical

Various portable respirators are of definite use in doing tracheotomy and in weaning the patient from the tank respirator They do not yet have the dependability or efficiency of the tank respirator.

The rocking bed seems to have proven of real value in making the convalescent patient independent of the respirator and shortening the transition period weeks or more.

Attempts at determination of chemical acidosis in our experience have not replaced careful clinical observation. Respiratory acidosis, as pointed out by Gamble,[21] gives opposite blood findings from those of metabolic acidosis and must be interpreted in the light of blood pH and compensatory mechanisms. To be significant, tests must be done on alveolar air, here impossible, or on arterial or capillary blood. We have obtained little value from them in individual treatment but they are very important for research.

The Los Angeles group, under the lead of Bower and Bennet, have designed a number of mechanical devices which careful physiologic studies indicate have much promise. Unfortunately they are only now becoming generally available and we have had no opportunity to use them.

They include *1.* The Bennet ventilation meter which aids in determining the setting of the respirator. It also shows when dangerous lessening of the tidal air indicates additional help by tracheotomy or the respirator. *2.* The positive pressure attachment which synchronizes through mask or tracheotomy with the negative pull of the respirator.

Some physiologists insist that in any case the true value of the negative pressure is the same as the algebraic sum of combined positive and negative pressure, but the Los Angeles group believe it definitely beneficial in more wide inflation of lungs and prevention or relief of pulmonary edema and atelectasis; and they found that usually a smaller total pressure is required and is better tolerated by the patient. Motley agrees that it is useful.

3. The flow sensitive breathing units appears to be valuable as for emergency use, especially tracheotomy, which can then be done out of the respirator. It also provides useful positive pressure for use in pulmonary edema. This is in accord with the ideas of Barach and the Committee on Inhalation Therapy.[21]

4. The cam adjustment appears to make a more comfortable and normal timing of the respiratory cycle.

The question of transportation of bulbar patients requires special consideration. Such patients are better treated in hospitals properly equipped, by teams properly trained, since some patients may first be lost while experience is being gained by a group to whom the problem is new. Travel and strain are so injurious to these patients except at the beginning of bulbar symptoms that they might do better if intelligently treated locally.

Polio in pregnancy is a special problem with a rather unfavorable experience until lately. The gravid uterus near term distends the abdomen, interferes with respiratory movements, embarrasses the heart and makes postural drainage poorly tolerated. If early preliminary tracheotomy is done, and the uterus emptied by Caesarean section, these patients seem to respond quite well.

CONCLUSIONS.

1. We believe that the idea that the serious effects in bulbar poliomyelitis are due to overwhelming infection of the vital centers is usually not justified.

2. Considered as a problem in secretional obstruction with anoxia and carbon dioxide accumulation, it lends itself to treatment that should be favorable in most cases.

3. This is a problem for a team of which the laryngologist should be a full member. He should see the patient at the first sign of difficulty in swallowing or of anoxia.

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PRACTICAL PROBLEMS IN THE USE OF CORTISONE AND ACTH.*

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Philadelphia, Pa.

I present this paper as an "acthologist." In attempting to present to a group of specialists in diseases of the ear, nose and throat the most important facts concerning cortisone and ACTH I was much perplexed, since importance is such a relative thing. What is of particular importance to you in this tremendous discovery of the effects of the pituitary adrenocorticotropic hormone (ACTH), or in an adrenal hormone as cortisone or the soon to be released compound F in various disease states? The smoke from this tremendous explosion in medicine has still not cleared away, but there are several points that I would like to discuss: First, these potent hormones produce a marked change in the entire body. They are master hormones. They are the hormones which control our resistance, which control our reactions to stresses of all types. They don't cure diseases, but they block the harmful reaction in many cases. For example, they produce a changed reaction to infection. Our rheumatoid arthritics very rarely have colds, because they are in a nonspecific hyperimmune state. They develop colds repeatedly and severely while on ACTH or cortisone, so we realize that one of the effects of these hormones is to change the mechanism of immunity. That is quite obvious since we have known physiologically for some time that the pituitary adrenocorticotropic hormone acting through its effect on stimulating the adrenal hormones (cortisone particularly) causes a lysis of lymphocytes, a release of antibodies and thus a decrease in the amount of immune bodies. Now that effect on resistance to infection, naturally, is important

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 12, 1951.

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to all of us in medicine. If we use cortisone, we must remember that a secondary infection can occur. Another effect which is brought to mind is the fact that the formation of fibroblasts, the formation of fibrosis in healing, is impeded by the action of these hormones. ACTH acts only through the adrenal by stimulating release of cortisone. Many times I will refer to cortisone meaning either hormone. The resistance to the effects of trauma is also changed in a similar way by a delay in healing of fibrous tissue. Conversely, that may have a beneficial effect in slowing the stenosis developed from large scar formations. This may have tremendous importance in surgery but has not yet been fully worked out. Actually, we may generalize by saying that the effect of these hormones in the conditions for which we are using them is much like that of pyribenzamine or similar antihistaminics in hay fever: it suppresses the reaction; it does not affect the cause. It is important also to realize the basic physiological changes induced, so that we do not use cortisone for a trivial process. It is really driving a tack with a sledge hammer. Here is a two-edged sword, one that is going to change our entire body physiology. Cortisone should not be used for trivial conditions, but should be saved as the "ace in the hole" for serious trouble.

I am not going into great detail regarding the complications, reactions and the methods of administration of cortisone and ACTH. These are enumerated quite well, I think, in the package circulars and in pamphlets that are easily available. The basic contraindications to the use of these hormones are worthy of consideration. On a patient in cardiac decompensation, the effect of cortisone or ACTH is an increased retention of sodium and fluid, and a loss of potassium. Edema is the most common complication; consequently, in cardiac decompensation we would have a great deal of difficulty from increasing the load. This is one of the great contraindications to cortisone. Another great contraindication is renal failure. We have seen some cases that have actually been thrown into uremia by the administration of ACTH or cortisone and, of course, again it may be due to the sodium retention, or it may be some actual effect on the kidney, the details of which are still not worked out, in fact, we do not know how these hormones act at the tissue level.

We are dealing with products which cause a definite immediate effect in many conditions It has been facetiously suggested that we should regroup diseases; those which respond to ACTH and cortisone and those which respond to antibiotics. As I said before, it doesn't cure any of them, but the self-limited conditions which are severe during their course may be greatly relieved We must regard these as agents for immediate relief, and often very dramatic relief, but they must be continued much as the diabetic is kept on insulin, to produce a consistent effect in chronic conditions. The rate of recurrence in anything but self-limited conditions, such as penicillin reactions or even acute rheumatic fever perhaps, is almost 100 per cent in our experience.

I will now discuss some of the local effects on the ear, nose and throat. In asthma, particularly severe and intractable asthma, status asthmaticus, ACTH has a dramatic alleviating effect. When gradually decreased to the so-called maintenance amount, starting with perhaps 100 mgm or 120 mgm. of ACTH a day, given in divided doses and then reduced, a patient can be pulled out of a severe status asthmaticus to sleep and breathe perfectly freely for the duration of the administration. The Hopkins group has been quite optimistic about the end-results on this and find very few relapses. In our own group at the University of Pennsylvania the relapse rate after the ACTH or cortisone is discontinued is quite high, so that we must continue a maintenance dose on cases serious enough to require it.

Effect on Hay Fever Randolph, of Northwestern, at the ACTH conference in Chicago last year reported the dramatic alleviating effect of ACTH on hay fever Again I want to emphasize the point that here we are driving a tack with a sledge hammer. We have other agents that relieve it, and I leave it to your own judgment whether ACTH should be used in hay fever.

Nasal Polyps. The Hopkins group have studied these with some detail and find that they are shrunken when the drugs are used in adequate amounts. The airways are opened, and there is a definitely alleviating action; however, in my small experience with nasal polyps, I find that surgery is far superior. When ACTH is discontinued, the polyps again return to their original size.

The question of lymphoid collections in the pharynx is rather important, I believe, since we know that these hormones have a definite shrinking action on lymphoid tissue generally. When there is marked lymphadenopathy the size of the nodes will decrease rather markedly when these hormones are used. Whether or not this is actually a beneficial effect remains to be proved. I think it suggested itself that it would be of benefit in trying to shrink the persistent lymphoid collections in the throat. Not being an otolaryngologist, I would not say definitely but it is my impression that here again we must think about the sledge hammer. I believe that local treatment should be far superior. Dr. Harvey and his group at Hopkins have reported that they found a moderate shrinking of lymphoid tissue and a change in the color of the lymphoid tissue with loss of the edema. After the drug is stopped these tissues return to their previous appearance, and there is often a rebound phenomenon in which the condition is temporarily even worse than it was originally.

There has been considerable confusion in the use of these hormones in infectious states. The use of ACTH in early tuberculosis has been recommended. It does break down the fibrosis which walls off pulmonary tuberculosis. In our own experience, sadly enough, tuberculosis can be lighted up quite severely by giving ACTH. We had one patient who had apparently an arrested T.B. On ACTH, given for rheumatoid arthritis, he had a severe flare-up with bilateral spread. The patient was naturally taken off the ACTH and is now improved, but it was rather alarming. A paper was read in Chicago at the second ACTH conference on Dec. 8 in which the authors reported using ACTH parenterally for tuberculosis of the larynx where there was marked granulation causing obstruction. In these cases they found that the effect of ACTH in shrinking the swelling and inflammation in granulation tissue seemed quite beneficial. They did not comment on what happened to the general state, and the follow-up on these cases was not of sufficient length to warrant any conclusion. I think tuberculosis is a definite contraindication to the use of the hormone.

There have been contradictory reports recently on what these hormones do to the coagulability of the blood. A recent paper in the *American Journal of Medicine* states that ACTH increases the coagulability of blood. They cite the danger of thrombophlebitis and pulmonary embolism; however, we have found an increased tendency to bleed from incision, and an increased tendency for nose bleed. As a typical example of this, I cite one of my rheumatoid arthritis cases who was given cortisone. Apparently it effected her immune response, and she got a severe cold, the first one she had had in four years. On blowing her nose she had a terrific nasal hemorrhage which took almost 24 hours to stop. The prothrombin time in these cases is not greatly changed and we do not see any other obvious hematological changes. On the other hand, however, it has been shown that postoperative shock is tremendously decreased. The recent reports on burns treated with ACTH have shown that epithelialization is not diminished as is the healing of fibrous tissue.

Another contraindication to ACTH or cortisone is psychic disturbance. A patient who is mentally on the ragged edge is a very poor risk for these hormones. We have had two very sad cases of arthritis which had had a psychotic episode in the past and who once more went into schizophrenia when cortisone or ACTH were administered.

The dramatic effects of these hormones in many conditions have been so widely accepted that I am adopting what might almost be called a negative approach. There can be no doubt that cortisone and ACTH are here to stay as part of our medical armamentarium. I am merely bringing out some of the things which we must keep in mind and be on the watch for when using these new agents.

Another complication that was seen in four of our patients was a breaking down of peptic ulcer. This does not affect you directly, but it is rather embarrassing to be treating a patient for a relatively minor condition with these hormones and then find that an ulcer, which had previously given few or no symptoms, proceeds to bleed or even perforate. Peptic ulcer is another contraindication to the use of the hormones.

Our experience with local therapy with cortisone has been somewhat limited in its scope. We have been using it particularly in rheumatic diseases. We put cortisone in the joint and we have not had any particular success with the drug locally. It may be that as compound F becomes more available, results will be different. The complications of cortisone therapy are very diverse. Occasionally we think we are seeing a new complication. I saw one a few days ago. Two consecutive patients complained of tinnitus. I thought here is another one! It just so happened that we had an otoscope handy and found impacted cerumen in both cases. We know that the secretion of the sebaceous glands is somewhat increased in many of these patients when we give these drugs. Apparently the increased secretion of wax was sufficient to produce an impaction of cerumen in ear canals that previously had not quite closed.

A practical problem in management aside from these contraindications, and one of the most important, is the manner of economics. We must realize that ACTH and cortisone, although they have been considerably reduced in price, are still very expensive drugs. This should make us pause and reflect on what is going to happen. In a patient with a severe chronic active state of arthritis, we must remember that the maintenance amounts of each one of these hormones is going to cost that patient for the drug alone about $20 a week over a long period. As yet we do not know how long.—possibly indefinitely like insulin; therefore, we must plan carefully. A psychological and physical let-down inevitably follows the stopping of these hormones. This is so severe that in chronic conditions we are doing the patients harm by giving them a course of the drug and then to stopping it; therefore, I repeat, the economic consideration must be brought out before starting. I think it is to the credit of Merck & Co. that although in November, 1949, it cost them $8,000.00 a gram to make cortisone they made it available to investigators at $300.00 a gram and just charged $7,000,000.00 to research. Today, just one year later, it sells at $28.00 a gram. This is American Industry! I think it is a wonderful contribution to medicine and deserving of commendation. Production is increasing and there is plenty of cortisone wherever needed. Cortisone and ACTH have been used for everything from snake bites to burns.

Probably in the field of ear, nose and throat surgery there is a relative lack of conditions where these hormones are really vitally needed. I have given you so many contraindications you probably wonder why you would ever use them. Again, on the credit column of the ledger for these hormones in the various states of rheumatic disease and in the various severe allergies, these drugs have a dramatic effect that has never been seen before, — a marked alleviating effect often within a few hours which is so marked that it has given us a tremendous new enthusiasm in medical research. It is up to us to evaluate these hormones objectively and to know with just what we are dealing. Again I say that cortisone and ACTH are valuable; they are here to stay; they cure practically nothing, but they relieve many conditions. We may have here a substance comparable to insulin in diabetes. Even though it has these marked side effects and many contraindications, when used carefully and in the right conditions it is a wonderful boon and a great advance in medicine.

BOOK REVIEW.

Rehabilitation of Hearing. A Guide to Modern Differential Diagnosis and Treatment of Hearing Disorders. By Victor L. Browd, M.D., Adjunct Professor of Otolaryngology, New York Polyclinic Medical School and Hospital; Associate Otolaryngologist, Midtown Hospital. New York, N. Y. 1951.

Within 38 pages are presented some facts dealing with the anatomy and physiology of the auditory mechanism differential diagnosis and therapy of hearing impairments. It is a clear exposition with several charts and amply illustrated by semidiagrammatic drawings. The author is to be congratulated for his ability to condense so many important subjects into such brief form. There is no doubt that the lectures for which the text forms a basis would be of considerably more interest. W. P. C.

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The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

June 1, 1951.

Audicon Models 400 and 415.

Manufacturer National Earphone Co , Inc., 20-22 Shipman St., Newark 2, N. J

Audivox Model Super 67.

Manufacturer. Audivox, Inc , 259 W 14th St , New York 11, N. Y.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co , 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.

Manufacturer The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co , Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Model R-2.

Manufacturer: The Microtone Co., Ford Parkway on the Mississippi, St. Paul 1, Minn.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C (See Micronic.)

Manufacturer Micronic Corp, 101 Tremont St., Boston 8, Mass.

(See Micronic.)

Silvertone Model 103BM.

Manufacturer National Hearing Aid Laboratories, 815 S Hill St., Los Angeles 14, Calif

Distributor Sears-Roebuck & Co 925 S Homan Ave., Chicago 7, Ill

Silvertone Model J-92

Manufacturer Sears Roebuck Co 925 S Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer Micronic Co 727 Atlantic Ave, Boston 11, Mass

Distributor Sears Roebuck Co., 925 S Homan Ave., Chicago 7, Ill.

Silvertone Model P-15

Manufacturer W E Johnston Mfg Co, 708W 40th St., Minneapolis, Minn.

Distributor Sears-Roebuck & Co 925 S Homan Ave., Chicago 7, Ill

Solo-Pak Model 99.

Manufacturer Solo-Pak Electronics Corp, Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer Sonotone Corp Elmsford, N Y

Superfonic Hearing Aid.

Manufacturer American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer Televox Mfg. Co, 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.

Manufacturer Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

Meeting: Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

Time and Place: Havana, Cuba, January, 1952. Dr. Jose Gros. For information write Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

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THE RESPIRATORY FUNCTION OF THE LARYNX *†

III — THE RELATION OF FIBRE SIZE TO FUNCTION IN THE RECURRENT LARYNGEAL NERVE

JOHN A MURTAGH, M D., and
CLARENCE J CAMPBELL, M D. (by invitation),
Hanover, N H

In a previous report[1] on the basis of measurements of chronaxie and observations on the activity of the glottis, we expressed the opinion that "the greater number of fibres in the recurrent laryngeal nerve are relatively large and supply adductor muscles," and again that "there are some few fibres supplying the abductor muscle which must be smaller and probably less heavily medullated Presumably these would be more readily injured by chemical or mechanical agents " With this in mind we have since examined histologically a number of "normal" recurrent laryngeal nerves in man and in the animal. The human nerves were obtained postmortem. We have also examined, when obtainable, involved recurrent nerves removed at autopsy in individuals who have presented the clinical picture of recurrent laryngeal abductor palsy and have attemped to evaluate the fibre population in the normal nerve and the existing variations from the normal in the paralyzed recurrent nerve.

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*Read at the Fifty fifth Annual Meeting of the American Laryngological Rhinological and Otological Society Inc. Atlantic City N J May 8, 1951

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METHOD.

The nerves were first placed in osmic acid solution (Department of Pathology, Hitchcock Hospital). This fixed and stained the myelin sheaths, and on these blackened sheaths all the measurements were made. When satisfactory sections were obtained, they were photographed. Enlargements were made and printed on bromide paper. The magnification was 720 diameters. Measurements were made of these prints by matching as nearly as possible the diameters of any particular fibre with one of a series of circles of given diameter etched on a plastic scale. None of the fibres was perfectly circular, so that an exact match was never possible. In consideration of this, a transparent scale was prepared with circles of diameters of 2, 4, 6, 8, 10 and 12 mm. The fibres were placed in one of six categories: 0 to 2, 2 to 4, 4 to 6, 6 to 8, 8 to 10 and 10 to 12 mm. Each millimeter on the enlarged photograph is equivalent to 1.39 micra. The analysis, however, is based on the measured diameters (in millimeters), remembering always that at no time was any attempt made to accurately measure any one fibre. All fibres are merely reported as falling within the limits of each category. The analysis is based on percentage of the whole in each group.

The procedure may be best understood by referring to Table I (R. L. N. 128 involved), which is the count on a nerve of a patient who died of carcinoma of the esophagus and who presented the clinical picture of recurrent laryngeal abductor palsy. The categories into which the fibres fall are reported as diameters 2, 4, 6, etc. In the vertical column on the left is the list of individual bundles which make up the nerve. The actual counts are recorded and totaled across to give the number of fibres in individual bundles and vertically to give the number of fibres in all bundles which fall into a particular category. The percentage representation of each category in the entire nerve is calculated and appears in the bottom horizontal row of Table I.

RESULTS.

As work progressed, it became more apparent that the recurrent laryngeal nerve is not a single fibre bundle as might

TABLE 1.

R L N 128 Left Involved I

DIAMETERS	2	4	6	8	10	12	TOTAL
AB_{10A}	5	3	13	51	4	0	76
AB_{10B}	48	226	174	52	5	0	505
AB_{11A}	0	5	14	29	35	8	91
AB_{11B}	7	34	49	56	3	0	149
AB_{11C}	0	16	73	38	44	0	171
AB_{12A}	17	40	25	6	0	0	88
AB_{12B}	3	4	13	0	0	1	21
AB_{12C}	2	59	45	37	16	5	164
AB_{13A}	103	204	163	85	10	1	566
AB_{13B}	47	136	107	50	3	0	343
AB_{13C}	77	216	136	52	10	0	491
TOTAL	309	943	812	456	130	15	2665
	11.6%	35.4%	30.5%	17.1%	4.9%	.56%	100.06%

be suggested by Fig. 5 of a previous paper.[1] There are invariably several 4 to 6, and frequently as many as 12 bundles of nerve fibres which appear to make up the whole nerve (see Fig. 1). The actual number of fibres in a single nerve will depend on the level at which the section is made. There are invariably more fibres peripherally than centrally as long as sections include the entire nerve (Eccles cited by Fulton).[2]

The actual percentage counts of various nerves that we have examined may be seen in Figs. 2 through 7. In Figs. 2, 3 and 4 are shown counts completed in "normal" human nerves. The striking similarity of nerves 1, 3 and 4 is brought out in Fig. 3. Human nerve No. 2 has more fibres in group 0 to 2 mm. than any of the other nerves examined. A possible explanation of this appears in Fig. 4 where the same nerve has been sectioned and counted at two levels. The central end of this nerve (A-50-26) shows some 33 per cent in category 0 to 2 mm., which compares reasonably well with count 0 to 2 in nerves 1, 3 and 4. In the peripheral end of human nerve A-50-26 there are some 1,500 more fibres in the total count than the central end of the same nerve, and the category 0 to 2 shows 48 per cent, which may be compared with 52 per cent in the same category in human No. 2. There exists, then, the possibility that the section of human nerve No. 2 may have been taken more peripherally than that of nerves Nos. 1, 3 and 4. The count in No. 2 is approximately 1,000 greater than that shown in No. 1, 3 or 4. This might be a further indication that this section was made more peripherally. It is also obvious that the counts in the first four human nerves is definitely smaller than counts in nerve A-50-26, which were

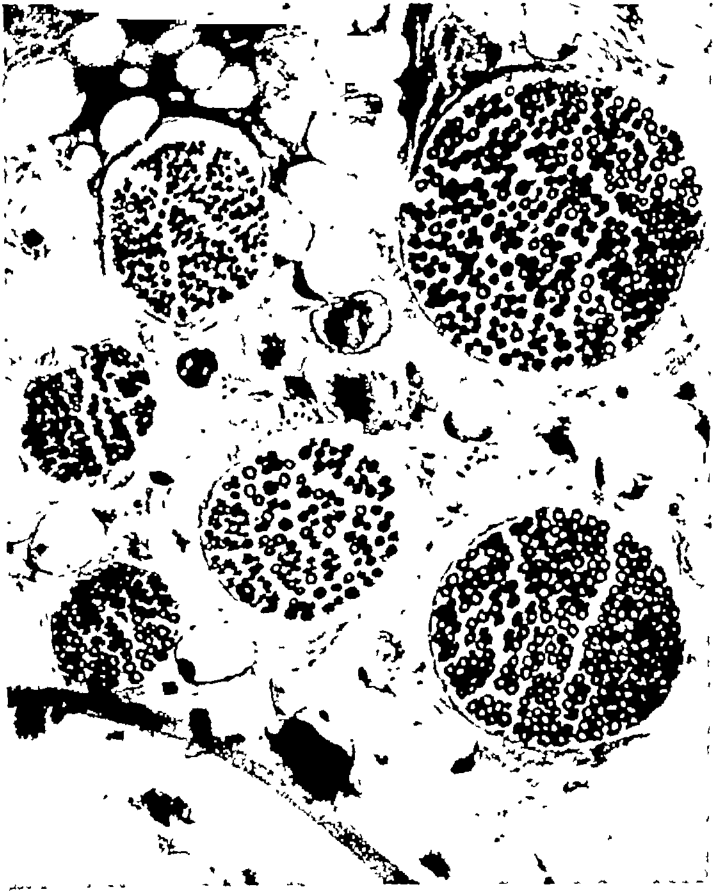

FIg. 1.

done at a later date. Every effort was made to include the entire nerve in all cases. Poor technique may be responsible for the low total counts shown or there may be a wide variation in the actual number of fibres. The histograms, based on percentage of total, show very similar distributions of fibre size in all nerves.

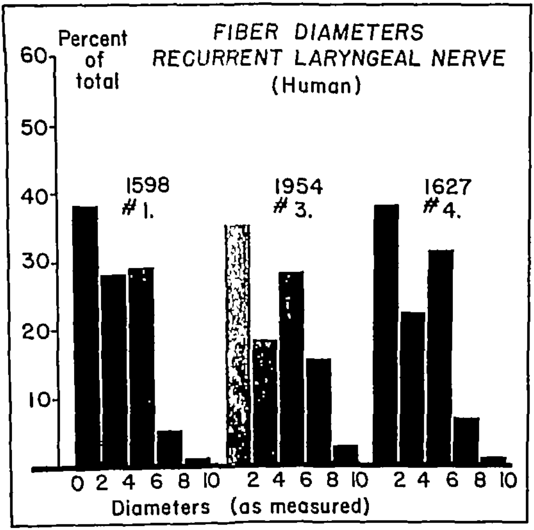

Fig. 1.

There appears to be a general similarity in recurrent laryngeal nerve of the human, the cat and the goat. There seems to be more of very large fibres in the goat than in either the cat or the human, and in each species we have one example where the percentage of smallest fibres is high. We have no record of the locus of these particular sections except in goat B. In this case the record shows that the "middle" of the nerve was the part sectioned. There is a definite possibility that in each of these cases the section was made more peripherally than was the case in the other counts.

So far we have only one nerve from a case of recurrent nerve palsy. The total fibre counts and the percentage representative at various levels in the nerve are shown, in Fig. 5. The striking thing in this figure is the low 0/0 representation of fibres in category 0 to 2. As the total count increases, the 0/0 in the 0 to 2 group goes up, but at all levels it is less than corresponding representations in the "normal" nerves. Since the laryngeal palsy was clinically an abductor paralysis, we think that the relative loss of small fibres is significant and bears out our previous contention that the abductor muscle is supplied by smaller and probably less heavily medullated fibres. More cases must, of course, be collected; it is folly to base any conclusion on a single instance.

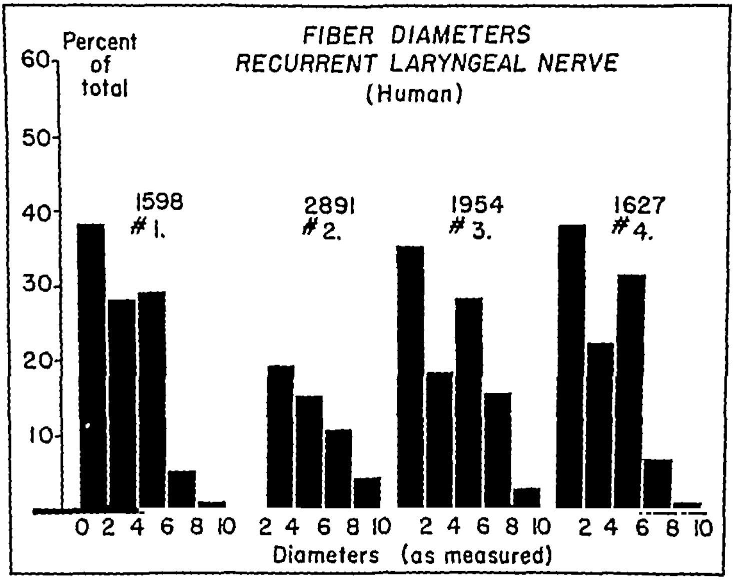

Fig. 3.

In some of the preparations examined but not reported here there was evidence of considerable and widespread degeneration of the myelin sheaths. This was considered to be postmortem degeneration. In one specimen reported, A-50-26, a patient who died of carcinoma of the colon, this same degeneration was observed, but the preparation was such a good one it was decided to count it anyway. Here the 0/0 counts agree with the other human nerves, and the degeneration has apparently affected all fibre groups, large and small, rather than selecting a single group, *i.e.*, 0 to 2 mm., as in the case of laryngeal paralysis.

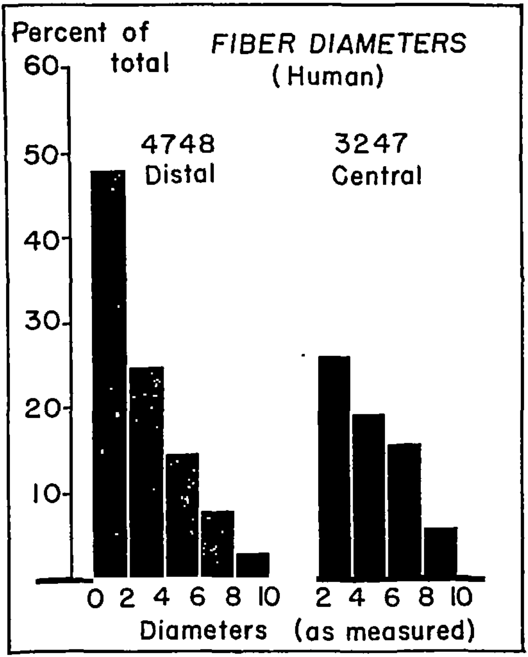

FIG. 4.

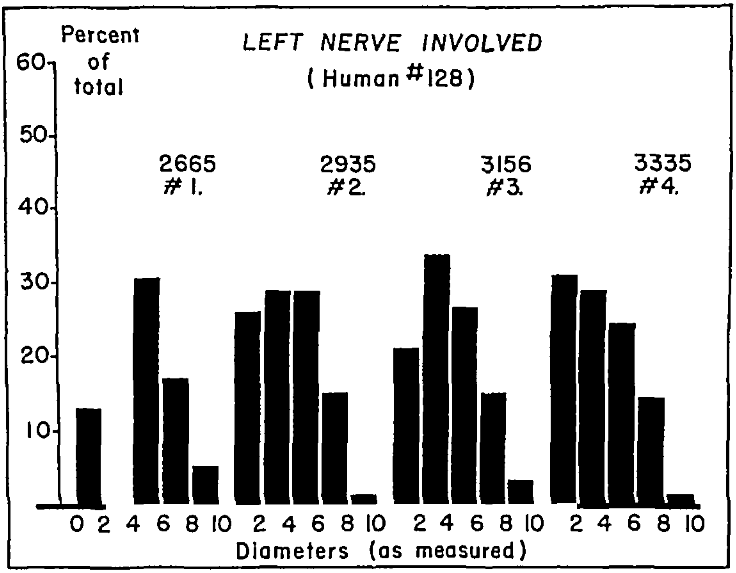
Percent of total
LEFT NERVE INVOLVED
(Human #128)
60
50
40
30
20
10
2665
# 1.
2935
#2.
3156
#3.
3335
#4.
0 2 4 6 8 10 2 4 6 8 10 2 4 6 8 10 2 4 6 8 10
Diameters (as measured)

Fig. 5.

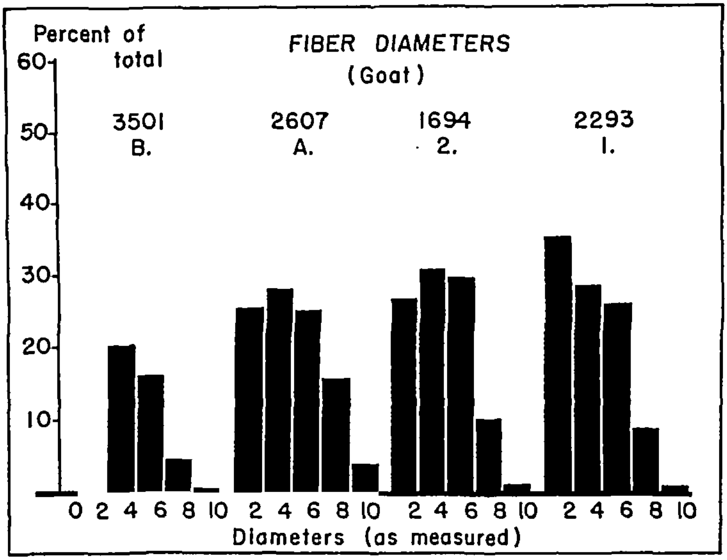
Percent of total
FIBER DIAMETERS
(Goat)
60
50
40
30
20
10
3501
B.
2607
A.
1694
2.
2293
1.
0 2 4 6 8 10 2 4 6 8 10 2 4 6 8 10 2 4 6 8 10
Diameters (as measured)

Fig. 6.

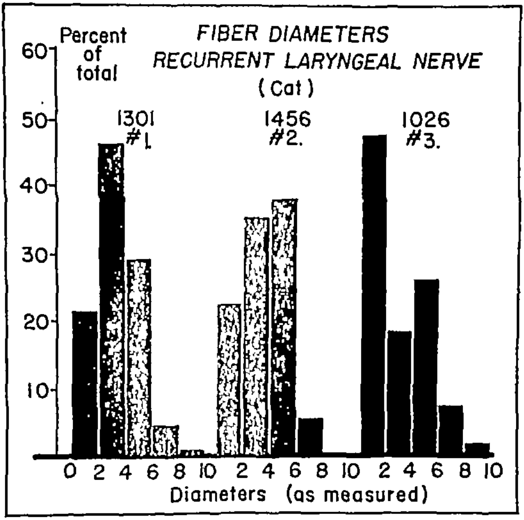

Fig. 7.

DISCUSSION.

The methods employed in this series limit the observations to medullated nerve fibres only. Postganglionic autonomic fibres, being nonmedullated, are not stained and hence are not seen. The recurrent laryngeal nerve is pre-eminently a motor nerve. There is no experimental evidence of sensory function.[3] It undoubtedly contains some afferent proprioceptive fibres. The properties of nerve fibres are conveniently tabulated by Grundfest.[4] Here the diameters of A fibres are given as varying between 1 and 20 micra. The A fibres only are considered to be associated with striated muscle and it is certain that it is with these fibres only that we are dealing. The diameters which we report fall within the group just over 2 micra to just under 17 micra. We have previously reported[1] our reasons for thinking that the smaller of these A fibres supply the abductor muscle of the larynx (*i.e.*, the posterior crico-arytenoideus).

SUMMARY.

The medullated fibres of the recurrent laryngeal nerve of three species, the human, the cat and the goat, have been counted and tabulated according to the size of the fibres.

The number of fibres and the size distribution varies somewhat, depending upon which level the section of the nerve is chosen.

In "normal" nerves, small fibres not over 2.8 micra in diameter, make up 30 to 40 per cent of the population.

In a case of recurrent nerve abductor paralysis, the population of the small fibres, less than 2.8 micra, is most affected by the degenerative process (11 to 31 per cent).

There is evidence (not statistically adequate) that the abductor muscle, as we have previously said, is supplied by the smaller fibres of the recurrent laryngeal nerve.

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CAUSES AND MANAGEMENT OF ABNORMAL BLEEDING FROM THE AIR PASSAGES ASSOCIATED WITH CERTAIN DISORDERS OF THE BLOOD.[*][†]

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We propose to discuss in this paper a few of the commoner disorders of the blood accompanied by a defective hemostatic mechanism, frequently manifested by almost intractable bleeding from the air passages. Abnormal bleeding wherever it may occur may result from: *a.* A local disturbance in hemostasis. *b.* A systemic disturbance in hemostasis. *c.* Combination of local and systemic defect.

The approach to the management of these disorders should begin by an attempt to trace the possible cause or causes responsible for the bleeding. Bleeding, whether open, or outside of the tissues, or into the tissues themselves, is the result of a temporary upset in the balance between the forces exerting a stress on the blood vessels and the degree to which these forces are offset by the hemostatic function of the body. These forces may originate outside (extrinsic) or inside (intrinsic) the body and, as readily surmised, vary a great deal in intensity, direction and type. In looking for causes of bleeding in any one person, it is well not to overlook the rôle played by these forces in precipitating and maintaining bleeding. Among some of the *intrinsic* forces is the blood pressure (arterial, venous, capillary), the movement of organs such as the tongue, pharynx, stomach and intestines, the expansion and contraction of the lungs. Aside from these, our bodies

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are exposed to *extrinsic* forces resulting from everyday activity, such as blows, infections, contact with chemical irritants, etc. Besides some degree of familiarity with these stress producing forces[1] knowledge of the main factors involved in the natural arrest of bleeding is helpful[2] in deciding the possible causes for *abnormal* bleeding.

The methods at our disposal to bring about systemic or local hemostasis largely mimic the steps that nature has provided for the spontaneous arrest of bleeding in the body. These are as follows:

a. External Compression. When blood is extravasated it accumulates in the tissues, raises the tissue pressure about the open vessel to the point of partially or wholly overcoming the pressure within the vessel. The application of compression to a bleeding wound by means of bandages, tampons, etc., is our way of duplicating this natural mechanism.

b. Contraction and Retraction of the Vascular Wall. When a vessel is severed it will often contract, twist and retract and thereby reduce blood flow. This we attempt to duplicate by, among other things, grasping the vessel with forceps, twisting and ligating it.

c. Formation of the hemostatic plug, into which go some of the formed elements of the blood, especially the platelets and the substances involved in the formation of the clot (prothrombin, fibrinogen, Ac-globulin). By means of transfusions, vitamin K and some plasma fractions, we attempt to supply the deficient or missing components of the natural hemostatic plug.

With this preliminary preparation, let us pass to the discussion of the cause and management of bleeding in patients with leucemia, hemophilia and hemorrhagic purpura.

Abnormal Bleeding in Leucemia. Epistaxis and bleeding from the gums are two common manifestations of acute leucemia, be it lymphoid, myeloid or monocytic, especially towards the latter phases of the disease. Hemorrhage is rarely observed in patients with chronic forms of leucemia. It is not uncommon for hematologists to have patients referred for study, by nose and throat surgeons or dentists, because of unanticipated bleeding during a tooth extraction, tonsillectomy or adenoidectomy.

Case 1: Mrs. W. K., age 28, during the early part of the Summer of 1949, had a severe sore throat which improved with antibiotic therapy. After much of the inflammation had subsided, tonsillectomy was undertaken. There was more than the usual bleeding during the operation, and sutures were required to stop it. The throat remained red and swollen, and the swelling spread to the gums of the upper teeth and soft palate. The patient developed a high temperature which could not be brought down by antibiotic therapy. It was then noticed that the leucocyte count fluctuated between 30,000 and 40,000 and that most of the cells were primitive monocytes. The platelets numbered 10,000 per cmm. A diagnosis of acute monocytic leucemia was made. The patient died approximately one month after the diagnosis was made and within two months after the tonsillectomy.

Several blood transfusions had been given soon after the tonsillectomy; they helped to reduce and eventually to arrest the bleeding. The early use of antibiotics had probably reduced the swelling somewhat and misled the surgeon into thinking he was dealing with a benign inflammatory disorder.

Difficulty in arresting bleeding after a tonsillectomy, accompanied by subsequent extension of the hemorrhagic or inflammatory process, should lead one to suspect the presence of a systemic disorder.

The cause of abnormal bleeding in acute leucemia has not been entirely elucidated. One or more of the following defects may account for it:

1. Thrombocytopenia.
2. Hypoprothrombinemia.
3. Leucemic infiltration of the vascular walls with consequent necrosis of the vessels.
4. Secondary infection, necrosis and sloughing of the tissues in parts such as the nose, mouth, throat and the lower gastrointestinal tract. The granulopenia that often complicates acute leucemia helps to make possible continuation of the infection.
5. Increase in anticoagulants in the blood.

A rational management of these patients should involve: *a.* Control of the leucemic process, *b.* blood transfusions, whenever possible, to be given by direct method, especially if a hemostatic effect is the main objective. *c.* Administration of vitamin K, 5 mg., by mouth or parenterally with hope of raising the plasma prothrombin, if this is reduced. *d.* Administration of vitamin C, 200 mg. daily, to help in the repair of the damaged vessels. *e.* Antibiotics, to combat any complicating infection. *f.* Diminution of exposure to any obvious stresses, thereby automatically diminishing tissue injury and additional bleeding.

All the above objectives can be fairly well carried out at present, with the exception of the first, *i.e.*, control of the leucemic process and the last, *i.e.*, correction of the excess of anticoagulants, when these are present in too high a concentration. Unfortunately we have little or nothing of significant value in delaying or arresting acute leucemia. Until effective methods of therapy for the disease itself have been developed, one must resort to the above measures for temporary control of its hemorrhagic complications. As in other systemic hemorrhagic disorders, it is wise to avoid the application of sutures or excessive or prolonged compression of the bleeding areas, in attempts at local control of the bleeding.

Even in the presence of some of the defects noted above, it is not uncommon to find little or no active bleeding in patients with moderate and severe degrees of thrombocytopenia and hypoprothrombinemia. Compensating forces or limited physical activity on the part of the patient may partially or completely offset the presence of the hemostatic defects.

It is not an uncommon experience to find that the local measures ordinarily proven to be effective in arresting bleeding in normal individuals may prove totally ineffective in patients with a systemic defect in the hemostatic mechanism. Indeed, some of these measures may actually aggravate or extend the bleeding. In general, it may be said that one should resort to only the mildest forms of local measures and depend more upon modifying the hemostatic mechanism systemically.

Compression of bleeding tissues in patients with systemic hemostatic defects should be exerted, not with the avowed *objective of driving the blood away from the superficial vessels* but with the intention of reducing the rate of bleeding so as to encourage the formation of a hemostatic plug Since, in patients with these defects, the plug forms slowly, the blood flow from the wound must be slowed, to permit the plug to be formed and acquire the needed firmness to resist the pressure of the blood in the vessels If excessive compression is placed on a wound, the part is rendered ischemic, and little or no internal plugging of the vessel results, when the compression is removed, the blood will again distend the vessels and flow out of their unplugged ends Aside from this, a continued ischemia may eventually lead to necrosis, sloughing and extension of the bleeding area

Chemical or thermal cauterization are frequently used in arresting bleeding from the nose and mouth. While this method in practiced hands is effective in patients with a normal hemostatic mechanism, it often complicates matters in those who have a systemic defect in hemostasis When using cautery, the physician relies on healing of the wound to replace the cauterized tissue *In patients with defective* hemostasis, there is a delay in the rate of healing so that even *though cauterization may temporarily arrest the bleeding,* when the cauterized skin or mucosa sloughs off, bleeding starts again, and usually from a wider surface than before cautery was applied This may lead perhaps to a second application of the cautery and temporary hemostasis, subsequent sloughing and a still wider wound In our clinic, we have had this experience so often that we have warned our colleagues against the use of either chemical or thermal cautery as a local hemostatic measure in these patients Again, it is preferable to depend upon systemic hemostatic measures than upon local ones It must be kept in mind that these systemic *measures themselves must be maintained throughout the* period of healing of the wound, in order to prevent recurrence of the hemorrhage before the wound has reached the stage when it can withstand the effect of motion and local trauma.

Abnormal Bleeding in Hemophilia. There are three clinical forms of hemophilia: *a.* Hereditary — when the disease is found in more than one generation of the same family. *b.* Congenital — when the disease dates from birth, with or without evidence of involvement of other members of the family. *c.* Acquired — when the disease appears spontaneously or as a complication of another disease in a patient who previously did not have the disorder. While the congenital and hereditary types are almost exclusively seen in men, acquired hemophilia may be seen in the male or female.

The defect responsible for the prolonged bleeding in hemophilia results in a delay in the rate of coagulation of the blood and this in turn results from an excess of antithromboplastin activity. Hemophilic blood has all coagulation factors in normal amount and quality, except for the excess of antithromboplastin. This excess slows the rate of transformation of prothrombin to thrombin and thereby the whole process of clotting. Since the rate of clotting is especially important in helping to arrest bleeding from the large vessels, hemophilics experience much of their bleeding in the deep tissues, especially the muscles. The tongue and the pharyngeal muscles are the favorite sites of hemophilic bleeding. These patients seldom bleed from superficial wounds of the oral, nasal or pharyngeal mucous membranes.

A word should be said at this time concerning the application of ligatures to bridge together defects of the skin and mucous membranes with the object of arresting bleeding. In most instances, especially in patients with hemophilia, suturing of parts like the tongue, the bed of a tooth or the tonsillar pillars is to be discouraged, since compression of the underlying tissues obtained by such measures, though appearing to arrest the bleeding from the surface, will often result only in driving the extravasated blood to the deeper layers of tissue which may become so distended as to crowd adjoining areas and interfere with its function.

Case 2: R. F., a known hemophilic, was admitted to Jefferson Hospital in extreme dyspnea. He could not close his mouth or lie down. The day before admission he had had a small laceration of the tongue, which had been sutured by his family physician The bleeding was thereby controlled but the patient noticed that his tongue was swelling until he could breathe only with difficulty and could not close his mouth. A laryngologist was consulted who advised aspirating the secretions from the mouth keeping the patient in a sitting up position and being ready to perform a tracheotomy if the swelling became worse. Five hundred cc. of fresh normal blood were given to the patient following the withdrawal of a similar amount from him Within four hours after the transfusion the patient s condition became stationary subsequently he began to improve and was discharged within four days most of the swelling having disappeared

The danger of asphyxia from hemorrhages in this area has been discussed in greater detail elsewhere[3] The patient reported by Endicott[4] died of asphyxia from a hematoma extending around the larynx, after the blood traveled down through the tissues from the original bleeding area, the bed of a recently extracted lower molar tooth The edges of the wound had been sutured after all attempts to check the bleeding by local and systemic measures had failed Rather than expose the patient to such hazards, our practice is to exert mild compression in the bleeding area to reduce, but not eliminate, blood flow and to replace blood loss daily, to maintain the blood pressure and allow sufficient time for healing to take place This may require as many as three weeks and 20 pints of blood Today, most fatalities in hemophilics rarely result from exsanguination, since the supplies of blood for replacement are now usually plentiful. Hemophilics frequently die from hemorrhage and the resulting compression of organs like the brain, the trachea and upper air passages The systemic control of hemophilic bleeding is by means of transfusion of blood from normal individuals. One cannot depend too much upon this measure, for its success hinges upon the severity of the hemophilia and the extent of the bleeding area Often one must be content with replacing the blood, protecting the wound and maintaining the circulation until healing eventually occurs Hemophilic wounds take from three to five times as long to heal as wounds in normal persons. During this healing period there may be many spurts of bleeding It is rare that a hemophilic wound does not bleed and stop bleeding several times before the wound heals definitively, and no further hemorrhage takes place

Abnormal Bleeding in Thrombocytopenic Purpura. In this form of purpura bleeding is manifested especially in the skin and mucous membranes. The mouth, nose and throat are favorite sites. The disturbance in hemostasis is related to the abnormally low number of platelets. This thrombocytopenia interferes with the formation of the platelet plug, which is among the most important steps in the permanent arrest of bleeding from the capillaries and venules where the pressure of the blood is comparatively low. Fortunately in thrombocytopenia there is little disturbance in hemostasis in wounds of large arteries, since vascular and intravascular factors, other than the platelets, share in the arrest of bleeding from these structures. It follows from this, that when accessible, relatively simple measures usually suffice to stop the bleeding locally, even though, often, the arrest may be temporary only. Slight pressure with a tampon having a well lubricated surface is often effective. Even more effective is delineating to the patient measures which will reduce or eliminate any possible stresses that may be exerted upon the bleeding area. For example, thrombocytopenic patients should be encouraged to sleep with the head raised well above the shoulders, to eat a soft diet and to avoid food that requires vigorous chewing or that has sharp edges that may cut the mucosa of the mouth and pharynx. They should be discouraged from undue straining during the passage of stools, for such straining will often precipitate an attack of epistaxis.

When compression is to be exerted upon the bleeding area, as mentioned before, it should be approximately of the order required to blanch the bed of a fingernail. Heavy compression applied with tight packs will result in devitalizing the compressed areas, necrosis of the tissue and more bleeding. The local use of so-called hemostatic substances often produce disappointing results. Frequently, the hemostatic effect really follows the application of pressure by the sponge or other body used as a vehicle to carry the hemostatic agent. If one must be tried, thrombin should be selected and applied on a thin sheet of synthetic absorbable gauze. The gauze holding the thrombin should be gently held against the bleeding sur-

face for about five minutes, so that it will "stick." A loose fitting pack well covered with a lubricant should then be placed over the gauze with the principal objective of maintaining it in contact with the mucosa and exerting light pressure As soon as convenient and practicable, an effort should be made at the correction of the thrombocytopenia. At present there are only three methods available for this purpose

1 Transfusion of fresh blood, preferably by the direct method This may be carried out by the use of silicon coated syringes In this manner the platelets of the donor are kept from being destroyed by contact with glass during the collection of the blood

2 Administration of Adrenocorticotropic Hormone or Cortisone In our hands, this method has been effective in only about one-third of the patients with thrombocytopenia In almost all instances, once the therapy is discontinued the thrombocytopenia returns

3 Splenectomy The decision to carry out this procedure depends upon a number of considerations that we cannot take up now Suffice it to say that except during an acute emergency, splenectomy should be reserved for those patients with chronic thrombocytopenia not secondary to infection, drugs or metastatic neoplasms, and whose bone marrow contains some megakaryocytes

Possible Methods of Enhancing Hemostasis The above remarks have dealt chiefly with well recognized methods of management of these forms of abnormal bleeding Let us proceed now to consider other possible methods for inducing hemostasis principally by enhancing the coagulability of the blood Normally the fluidity and coagulability of the blood are maintained by a balance between the amount of procoagulants and anticoagulants in the blood[5] There are, generally speaking, two ways of increasing the fluidity of the blood

1 Increasing the amount of anticoagulants or enhancing the effect of the natural anticoagulants in the blood, by adding to it substances like heparin. This is now fairly commonly practiced in the management of thromboembolic disease

2. Reducing the amount of a procoagulant and thereby enhancing the action of the undisturbed anticoagulants. This is what is done when a decoagulant substance like dicumarol or other prothrombin reducing agents are employed in the management of thromboembolic disease.

What measures then should be taken if the ultimate objective desired is the arrest of hemorrhage by increasing the coagulability of the blood? Logically two approaches are open:

a. Attempt to increase the procoagulants in the blood, especially those that have to do with the initiation of clotting (*e.g.*, platelets), the acceleration of clotting (Ac-globulin), or the substrate of clotting (prothrombin, fibrinogen). So far there are no agents available (with the exception of vitamin K for prothrombin and cortisone and ACTH for platelets) that can be relied upon for this purpose.

b. Reduce the natural circulating anticoagulants and thereby enhance the effect of the natural procoagulants, even when the procoagulants themselves have been reduced by disease, as for example, during the hypoprothrombinemia of hepatic cirrhosis, or in the thrombocytopenia of acute leucemia. This approach would seem to be promising of fruitful results, since the stability of circulating blood and the delay in the inception of clotting is related to the concentration of these anticoagulants (antithromboplastin, antithrombin). Attempts to offset the action of these anticoagulants should render the blood hypercoagulable and to a certain extent enhance the hemostatic mechanism. This applies especially to those instances when the deficiency in procoagulants cannot be corrected by the available means (hypoprothrombinemia refractory to vitamin K, thrombocytopenia of myelophthisic anemia).

Stomatologists and nose and throat surgeons are faced with the problem of controlling hemorrhage much more frequently than that of combating thromboembolic disease. We have been making tentative trials *ex vivo* with agents designed to reduce plasma antithromboplastin activity. Some of these agents reduce this activity to such an extent that the plasma clots rapidly after addition of small amounts of thromboplastin, even in the absence of calcium. Hemophilic plasma which has an excess of this activity can be made to clot in a shorter time than normal plasma, after being exposed to these agents. It is important to stress that these agents, if and when their use becomes practicable, should be employed with great care and discretion and with a full realization of their intrinsic danger. The field of usefulness for them should be in that group of patients whose excessive bleeding results from a deficiency in prothrombin, platelets or Ac-globulin, but whose anticoagulants (antithromboplastin, antithrombin) are in normal or excessive amount It may be possible by this means to

1. Reduce blood loss during operations in individuals with a normal hemostatic mechanism

2. Prevent *excessive blood loss in those with a defective* hemostatic mechanism, though the defect may not be caused by any changes in the blood

3. Reduce or prevent excessive blood loss in patients with defective hemostasis caused by deficiencies in procoagulants *or excess of anticoagulants The neutralization of the natural* circulating blood anticoagulants should leave the natural procoagulants of the blood in a position to exert their action more effectively and thereby promote rapid hemostasis.

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MEDICAL AND CYTOLOGICAL DIAGNOSIS OF BRONCHIOGENIC CARCINOMA.*

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It is the intention of this paper to summarize rather briefly the current conclusions regarding bronchiogenic carcinoma as these affect the practice of the clinician. Where new findings are accumulated so rapidly, oftentimes from the point of view of highly technical specialties, a clinical summary is sometimes helpful in reminding us that bronchiogenic carcinoma remains basically a clinical problem.

It is rather strange to find that one of the most discussed questions concerning this disease, and one of the most widely discussed questions in medicine, is statistical. Has there actually been a real increase in the incidence of bronchiogenic carcinoma? The apparent increase is overwhelming. Not too long ago bronchiogenic carcinoma was universally regarded as a rare disease. Today it is a major problem in every chest clinic in the United States. According to some autopsy statistics, there has been a fortyfold increase since 1900. Bronchiogenic carcinoma is now listed as the second most common malignancy of the male sex. We have been interested in carcinoma statistics at the University of California for over a year, and we believe that pulmonary malignancy is actually first in frequency. It is possible that the statistics of the Bureau of Public Health, when they are tabulated, will show bronchiogenic carcinoma the most common form of malignancy in males in this past year, 1950. Deaths from the disease may reach 15,000.

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For a long time many of the people concerned with this statistical problem insisted that the yearly increase in this particular form of malignancy was an illusion, attributable to better diagnosis and so forth. This theory is increasingly hard to maintain. More and more people are accepting the fact of a real increase and are looking around for the reason.

Etiology. From one point of view, the interest in possible etiologic agents for bronchiogenic carcinoma has been quite fruitful. Dozens of such substances have been proven in the laboratory, and several industrial situations, most notably pitch-blende mining and chrome refining, have been proven to be carcinogenic. Such industrial hazards, however, are not really of much significance so far as the question of increased incidence is concerned. Anyone looking over a representative collection of case histories will very quickly be convinced that in not one case out of a hundred is there the remotest possibility that the disease results from some specific industrial situation. With very minor exceptions, bronchiogenic carcinoma is not an industrial hazard.

There have been many attempts to find some factor in the modern environment that might account for the increasing incidence in bronchiogenic carcinoma. Most work has been done on dust which contains petroleum products and on tobacco smoke. Graham, Oxnard and many others have felt that cigarette smoking is a definite factor in the increased incidence. Wynder and Graham recently published a study which was quite impressive. In a comparison of almost 700 patients with bronchiogenic carcinoma with a group of controls of a similar age and sex distribution, they found a significantly higher percentage of steady and heavy cigarette smokers among the patients with carcinoma. A similar study is in progress at the California State Department of Health in which everyone diagnosed as having bronchiogenic carcinoma is being very carefully questioned by trained investigators concerning their smoking habits and other possible etiologic factors. The survey is not complete, but I understand that it looks very much as though this study will bear out the conclusions of Wynder and Graham.

Material. We became interested some time ago in the problem of determining the actual clinical status of bronchiogenic carcinoma. There have been several large and painstaking collections of case histories reported in recent years, but it seemed to us that perhaps these studies did not portray the general situation, because the material was drawn exclusively from teaching institutions and special chest clinics, or cancer clinics. To avoid this, we went back to autopsy material collected from 19 California hospitals which were selected to give us some kind of representative cross-section. We collected 1,070 autopsy reports, and much that I have to say is drawn from this material. We have tried to find out how the disease appeared to the medical profession at large, rather than how it appears to those who are especially interested in cancer.

Symptoms. We did not find anything especially new so far as symptomatology is concerned. The important thing to notice is that bronchiogenic carcinoma is not characterized by particular definite symptoms, but by a group of symptoms. Whenever any of these occur in a person over 30 or 35 years of age, especially of the male sex, they must be investigated and reinvestigated. Cough was, as usual, reported most frequently — a total of 75 per cent. These figures depend ultimately upon the observations of medical students and interne records. We believe that if these patients had been carefully questioned, a history of cough would have been elicited in almost 100 per cent, although in many cases it would be a morning cough not much more severe than that of millions of healthy people. Where, in the interest of our cytological studies, we have pressed the patients to produce sputum, we have found that they can almost invariably produce enough for our needs. The rest of the symptoms reported are those that are commonly found. Weight loss, chest pain, hemoptysis, shortness of breath, hoarseness, complaints attributable to the central nervous system, metastatic masses — these occur over and over in case history analyses. Eighty-five per cent of these patients had some kind of symptom referrable to the chest cage, although it was not necessarily the most prominent complaint.

Physical Findings. The most significant physical findings are tracheal deviation, pleural fluid, wheeze and clubbing of the extremities. A unilateral wheeze is a very significant finding. If it occurs in a male over 35 years of age the only safe procedure is to call it bronchiogenic carcinoma, and then try to rule out this diagnosis. Nineteen per cent of these case histories showed some pleural fluid detectable by physical examination. In the past, pleural fluid has been taken to prove inoperability. It probably does prove inoperability when the fluid is bloody or infected, but in over half of these patients the fluid was clear. Any lung infection can produce clear pleural fluid without directly involving the pleura. This question must be finally answered by the surgeons, but it seems hasty to conclude that pleural fluid indicates inoperability without trying to find out if the pleura really is involved. It should be pointed out that pleural fluid can be almost as suggestive as a unilateral wheeze to a diagnosis of bronchiogenic carcinoma. It is very unusual to see a primary tuberculous pleural effusion in a person over 40 years of age.

Clubbing is also a suggestive finding. It occurred in 9 per cent of the patients in this series. While this deformity can be associated with almost any abnormal pulmonary process, it usually appears only after the pulmonary pathology has become chronic. When clubbing is observed, and there is no history of long standing pathology, bronchiogenic carcinoma must be the first consideration in diagnosis.

In this series of cases, 438 patients, or about 45 per cent of the total studied, had evidence of metastatic lesions when they first presented themselves for examination, or developed such evidence very shortly thereafter. It is this group which is very poorly represented in statistics from teaching institutions or clinics. When the primary condition is recognized as bronchiogenic carcinoma, there is obviously no point in referring such patients for special attention. This group emphasizes the fact that surgery alone can never be a very satisfactory answer to pulmonary malignancy.

X-ray Findings. In this study, adequate Roentgen reports were obtained for only 889 patients. We hear a good deal about negative X-ray reports in bronchiogenic carcinoma. We had 27 such instances in this series. It is probably well to note that this situation is uncommon, but also to recognize that it exists. For the rest, 65 per cent of these X-rays were read as "suggestive mass," and the balance, 33 per cent, had evidence of atelectasis. These findings were not uncomplicated, however. Pleural fluid tended to obscure the basic condition, and "pneumonia" was very common, 34 per cent. No disease masquerades as something else more commonly than bronchiogenic carcinoma, and it most frequently appears in the guise of pneumonia. Perhaps no single procedure would be more effective against bronchiogenic carcinoma than adopting the custom of X-raying pneumonias after the usual course of antibiotics.

Bronchoscopy. We were rather surprised to find that in these 1,070 cases, only 397 were bronchoscoped. Since this study extended over a period of years, it is probable that at the present time a much greater percentage of bronchiogenic carcinomas are bronchoscoped. The results of these 397 bronchoscopies do not seem to us far out of line with our own experience. In 174 cases, the bronchoscopy was reported as completely negative. In 97, positive evidence of malignancy was obtained; in the balance, comprising one-third of the patients bronchoscoped, some kind of inconclusive evidence was observed. The positive results obtained in this series are perhaps a little low. Herbut and Clerf obtained diagnostic biopsy material in about 34 per cent of 307 patients. We doubt, however, that the 60 to 75 per cent of positive findings which have occasionally been reported can be maintained in a large series of unselected patients. No matter how many mirrors are used, or what techniques for biopsy beyond the field of vision are devised, it looks as though less than half of our bronchiogenic carcinomas are susceptible to bronchoscopic diagnosis. Also, while it is probably not true to say that a positive bronchoscopy indicates inoperability, it is obvious that those tumors most amenable to surgery frequently lie outside the range of the bronchoscopist's vision.

Surgical Results There is a large question as to whether bronchiogenic carcinoma can ordinarily be considered even potentially operable We have been able to follow the first 241 cases of bronchiogenic carcinoma diagnosed by our cytological studies. Of this group, 124 were deemed inoperable at diagnosis The remaining 117 were explored, with five operative deaths. Of these 112 patients, 48 were found to be inoperable when the chest cage was opened; the remaining 64 were resected To our certain knowledge, there was recurrence within a very short period in 41 of the 64 resections; of the 23 presumed survivals seven disappeared shortly after the operation Nine others had a recurrence more than six months after the operation Of the original 241 cases of bronchiogenic carcinoma, only seven are known to be alive 11 months after the operation

It is easy to despair when our best efforts can produce less than a 3 per cent survival rate, and these by no means out of danger. We at one time contemplated a paper on the hopelessness of bronchiogenic carcinoma, but, on the other hand, patients with bronchiogenic carcinoma *have* survived for five and even 10 years after their pneumonectomy We must do the best we can with the techniques that are available. Even if we cannot be sure of these seven survivals out of 241 diagnoses, we can reasonably hope that two to four of these patients will become five-year "cures." We must accept this 1 to 2 per cent survival rate and try to improve upon it. It is obvious that so long as surgery is our only answer to bronchiogenic carcinoma, the cure rate will be low. Probably well over one-half of such growths have metastasized, or otherwise become inoperable, before they produce even moderate symptoms; but if we cannot, with surgery, save 50 per cent, we can perhaps save 20 per cent. A 20 per cent survival rate under the circumstances would be a substantial achievement, and it does not seem unobtainable

Cytologic Diagnosis. We became interested, about four years ago, in the staining and smearing techniques with which Papanicolaou and Traut were so successful in the diagnosis of uterine and cervical cancer. Since that time we have examined specimens of sputum and bronchoscopic smears from over 2,500 patients with chest pathology. We are still using the procedure substantially as described in the reports of these men. We have had to modify some of their findings, of course, in the light of our experience with bronchial secretions. In particular, we have found it necessary to insist upon five separate specimens of sputum before we can speak with any assurance, but the method works; it works on early cases, and it works on tumors beyond the effective range of the bronchoscope.

Many pathologists were doubtful of this technique, apparently believing that we attempted to make a diagnosis upon the basis of a single cancer cell. We do not do that. We analyze groups of cells according to several criteria of malignancy. In many respects the technique is more accurate than the examination of sections of tissue by which the pathologist arrives at a diagnosis. In the latter instance, the tissue comes from the fixative shrunken from four to 10 times. The paraffin fixation and staining often further distort the specimen. In the examination of sputum, on the other hand, the cells do not undergo appreciable shrinkage if the specimen is properly handled. Generally the cells we see under the microscope are about eight times the size of those seen in the ordinary H and E preparation. Further, minute details of appearance are retained by the simple process of smearing and staining developed by Papanicolaou and Traut. As a consequence we have been able to establish as criteria of malignancy details that are not even observable in the usual biopsy specimen. Let me say again, however, that we do not depend upon a single criterion. We look for excessive variation in cell size, prominent nuclei, variation in the size and shape of the nuclei, jagged protrusions of the nuclei, irregular condensations of chromatin, the smearing of the chromatin, and so forth. We do not rely on any single one of these appearances. Our first object has been to establish the reliability of the technique; improved sensitivity has been the secondary consideration. We believe that cytologic diagnosis is morphological, if the diagnostician is sufficiently experienced.

REPRESENATIVE CASE HISTORIES.

Case 1: A 61-year-old baker entered the hospital in June, 1948, because of a density in the left lung discovered by a routine X-ray. He had a history of cough for two years previously, which was productive. On several occasions his sputum had been blood-streaked. A positive cytologic diagnosis was made without much difficulty, and within a week of entry a bronchoscopic examination revealed a sanguineous exudate in the left lower bronchus A left pneumonectomy was performed two days later and an epidermoid carcinoma was discovered. The patient is well and active three years after the operation.

Case 2: A 47-year-old carpenter entered the hospital on June 2, 1949, complaining of pain in the anterior portion of his left chest, which had been constant for four months Four months earlier, when the pain had begun, a chest X-ray showed a faint shadow in the left upper lung field, and at this time there had been both cough and sputum. At entry into the hospital, the lung shadow was dense, but the cough had disappeared. Tentative diagnosis was tuberculosis, with carcinoma to be ruled out. Eight days after admission malignant cells were found in the sputum. A bronchoscopic examination revealed a deformed carina and purulent exudate in the left bronchus. Malignant cells were also demonstrated in this exudate. The left upper lobe and lingula of the lower lobe were removed. The patient is well at this date.

Case 3: A 70-year-old cook entered the hospital on Dec. 19, 1949, with a diagnosis of pneumonia. An inflammatory process in the right lower lung field cleared with antibiotics Sputum revealed cells consistent for bronchiogenic carcinoma, although the clinical diagnosis was bronchiectasis or lung obscess A segmental resection was performed, and pathological laboratory reported only bronchiectasis and inflammatory changes. Further sputum studies were undertaken; malignant cells were still present. The patient died postoperatively. At postmortem a small carcinoma, not larger than a quarter inch in diameter, was discovered, lying just above the resected portion of the lung.

Case 4: A 66-year-old man had had one sputum specimen positive for acid-fast bacilli and was sent to a sanatorium. An X-ray revealed an area of cavitation. Differential diagnosis was tuberculous abscess or posslbly a coccidioidal abscess. Sputum studies revealed malignant cells. A pneumonectomy revealed a tumor lying about the abscess cavity. The patient developed a bleeding duodenal ulcer postoperatively, and died. Postmortem confirmed the finding of coexistent tuberculosis and malignancy.

It is perhaps worth notice that we have collected to date 131 cases of coexistence of pulmonary tuberculosis and bronchiogenic carcinoma. We believe that cytology is revealing carcinoma in many cases of tuberculosis in which, in the past, the malignancy would have been overlooked.

Case 5: A man of 50 years had repeated episodes of hemoptysis, but his X-rays were reported as negative. Studies of sputum samples revealed, however, malignant cells. Bronchoscopy was negative at first, but we continued to demonstrate malignant cells, and repeat bronchoscopy showed an inoperable tumor lying at the carina.

This last case report brings up an interesting situation which has already occurred several times at the San Francisco Hospital. What is to be done when the sputum examination reveals malignancy, but there is no way of determining its location? It is a very difficult position for the physician. In most instances lipiodol will help to locate the tumor body, but occasionally we must continue to utilize special X-ray techniques and bronchoscopy and wait for developments. This is fortunately a rather rare occurrence.

On the whole, we are encouraged with the results that we have obtained with cytology. The number of patients that have been examined by this method is at the present time considerably greater than 2,500, and we have already reported the results from the first 2,000 so examined. In this group, 241 positive diagnoses were confirmed by morphologic methods other than cytology. In another 120 we were able to confirm a diagnosis of metastatic lung cancer. In an additional 121 we made positive findings, but other morphologic evidence was not obtainable.

Of the 241 bronchiogenic carcinomas positively established in this group, the first morphologic diagnosis was made by means of cytology in 47 per cent. In an additional 46 per cent, cytologic diagnosis was made concurrently with diagnosis by some other morphologic technique. Thus, a positive cytologic diagnosis was achieved in 93 per cent of the 241 cases. As has been said, this degree of sensitivity depends upon our securing a minimum of five sputum samples. Where only one is available, we have reported an accuracy of about 55 per cent. Actually, we believe that even one sample is apt to be more accurate than this figure indicates; the overall figures include a good many samples shipped to us from other places, in which the fixing technique was unsatisfactory. We have been able to detect about 70 per cent of the bronchiogenic carcinomas at San Francisco Hospital on the basis of the first sputum sample submitted.

Summary. The clinical problem of bronchiogenic carcinoma is the problem of early diagnosis. On the basis of 6,000 sputum specimens examined, we believe that cytology can contribute materially to that end. It is a simple technique that can be performed routinely, and repeated indefinitely without serious inconvenience to the patient. Cytologic diagnosis is morphologic, and an accuracy of over 90 per cent can be anticipated if five sputum samples are collected. In our experience, virtually every patient can raise sufficient sputum if the physician insists upon it. Early morning sputum is most apt to reveal significant findings.

In our experience with 2 000 patients, 241 positive diagnoses of bronchiogenic carcinoma were made. Even with the help of cytology, the survival rate in this group has been very small; however, most of these cases were found among the hospital population. It seems that by the time patients are driven to accept hospitalization for the symptoms of bronchiogenic carcinoma, practically all of them are hopeless. In our studies, we found that the average patient with bronchiogenic carcinoma survived only six weeks after admission. If we are to achieve a material improvement in the survival rate of this disease, we must make our diagnosis before symptoms become alarming.

Dr. Brodie Stephens has suggested that every male over 35 years of age be routinely X-rayed yearly. We concur. But perhaps cytology can contribute significantly to the problem as X-ray. We believe that it should be a routine procedure for every person over 35 who has cough, sputum, inexplicable fatigue, or any other complaint conceivably to be referred to an underlying malignancy. If we can make this routine practice, undertaken long before hospitalization is considered, we can detect a considerable percentage of lung cancers while they are still operable.

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Deafness beginning at a very early stage and progressing rapidly, audiometrically shown to be a combination of impaired sound-conduction and sound-perception, strongly points to a tuberculous affection of the ear.

In the beginning, nerve deafness might even be more pronounced than middle-ear deafness, probably due to a tuberculotoxic irritation of the labyrinth. With the further progress of necrosis of the tympanic membrane and the ossicles, however, impairment of sound-conduction grows decidedly worse, often to the extent of total deafness. Close attention to all symptoms suspicious of tuberculosis has become a very important task today, because the earlier streptomycin treatment is begun the better is the prognosis of the case.

In about 50 per cent of our cases the tubercle bacilli cannot be found, and our diagnosis is based entirely upon the clinical symptoms. Treatment with streptomycin is instituted immediately. U. Russi claims that the best results are obtained by the local application of streptomycin. For this purpose the external auditory canal, the perforation in the tympanic membrane, and the tympanic cavity are cleaned as well as possible. A 10 per cent solution of streptomycin in water is then sprayed as an aerosol into the middle ear and, if possible, into other parts of the pneumatic system, at least once a day over a period of weeks, or, perhaps, even months. Most resistant to this therapy are those cases of tuberculous otitis media in which extensively pneumatized mastoid processes are found.

I find that the best therapeutic results in exudative tuberculosis of the middle ear are obtained by combining the local administration of streptomycin with the parenteral administration. The antibiotic injected intramuscularly is taken by the blood at least to the edges of the tuberculous foci which lie in the mucous membrane and in the bone of the mastoid. By this procedure further spreading of the tuberculous process into deeper structures may be prevented. For this purpose a total of at least 60 gm. of streptomycin in daily maximal doses of 24 mg. per kg. body-weight should be administered.

On the other hand, the locally applied streptomycin directly attacks the tubercle bacilli in the ulcerations and granulations in the mucous membrane of the tympanic cavity.

If the tubercle bacilli are resistant to streptomycin, treatment with a 20 per cent para-amino-salicylic acid solution should be attempted. By local application of para amino-salicylic acid combined with marbadal (a thiocarbamide preparation) Schuermann has achieved cessation of ear secretion, fading of the inflammation of the eardrum, clearing up of the mastoid process with new calcification of the bone, and an increase in hearing distance.

U. Russi is now working on a thorough statistical evaluation of his cases. It is possible that when he has finished this work my statements today may have to be corrected. By the local administration, as described above, cessation of middle ear suppuration, healing of the eardrum perforation, and a marked improvement of hearing was achieved in only five cases of specific otitis although 77 cases of tuberculous otitis media were observed from their onset and streptomycin treatment was instituted immediately upon perforation of the eardrum. Herewith is a brief report of one of these rare cases.

Case 1: B. M. The patient is a 23-year-old girl with a cured pulmonary tuberculosis (see Fig. 2) and onset of a purulent otitis media in May, 1947. Tubercle bacilli were found in the pus and in the adenoid vegetations removed in August, 1947. On Aug. 18, 1947, the left ear showed a markedly thickened, yellowish red colored tympanic membrane with a large central perforation. In the granulating mucosa of the tympanic cavity there were a few caseous foci. Hearing distance in the affected ear for whisper was 15 cm., for conversational voice, 150 cm. Local therapy with streptomycin was begun immediately. Within 10 days the ear secretion ceased and within five weeks the hearing distance for whisper increased to 500 cm. At this time the eardrum was dry and thickened by scar tissue. There was still one small perforation in the anterior lower segment. Nine weeks after beginning of the treatment the hearing distance for whisper was 600 cm. The perforation in the scar-thickened eardrum was closed. The streptomycin treatment was discontinued. In June, 1948, January, 1949, August, 1949, and January, 1950, short recurrences of acute inflammation without perforations occurred in the right ear, accompanied by a decrease to 20 to 30 cm. in the hearing of a whisper with each recurrence. On July 14, 1950, the tympanic membrane was markedly thickened, but not irritated. The hearing distance on the left ear for whisper was 200 cm. and for conversational voice, 800 cm. The audiogram showed an average air-conduction loss of 25 db over the entire frequency range. The patient has been under constant control since then with no recurrence.

As I have pointed out, such healing is very rare in our experience but by the local application of streptomycin we have achieved definite improvements in 94 of 101 cases of tuberculous otitis media. In these cases ear secretion ceased within one to three weeks. In about 30 per cent of all cases there is a recurrence of purulent otitis, partly specific and partly nonspecific in nature. It can also be arrested promptly by a second and third local treatment with streptomycin. Cessation of the secretion probably parallels a certain degree of stabilization of the disease process. The eardrum perforations, therefore, usually do not expand, and destruction of the ossicles ceases. In our 101 cases we found no further complications in the mastoid processes or in the endocranium. Regarding the loss of hearing, however, once such a loss has manifested itself, the success of the streptomycin treatment in this large group of merely improved cases shows considerably less satisfying results. Disregarding a few slight

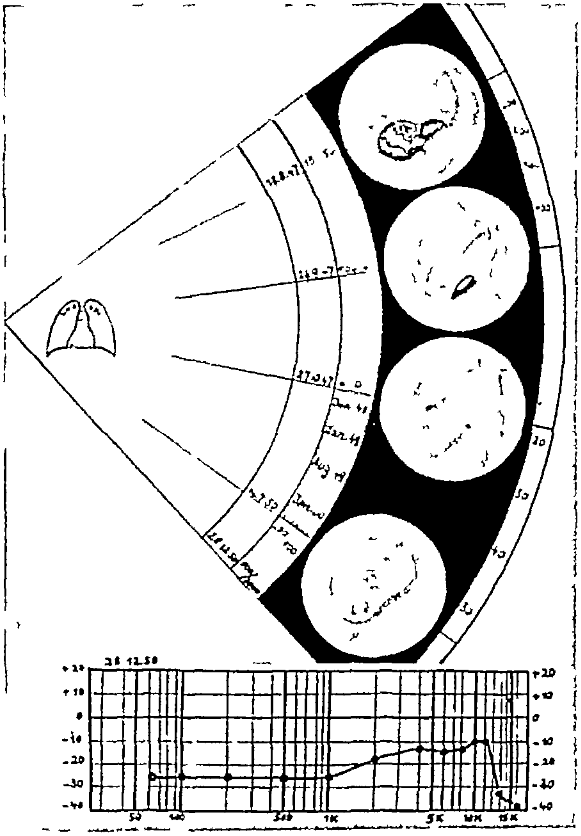


Fig 2 Twenty-three-year-old girl with pulmonary tuberculosis Healing of otitis media tuberculosa dextra after local therapy with streptomycin.

improvements of hearing the hearing loss found at the onset of the treatment usually remains unchanged. Remembering, on the other hand, from experiences of the past, the stabilization of the process damaging the hearing, or the preservation of the intact remnants of hearing, must be considered successful therapy. The success of the streptomycin teatment in these merely improved cases may be challenged by a non-specific new infection of the open middle ear or by a recurrence of the old tuberculosis.

It is a pity that we have no histological slides of tuberculosis of the middle ear treated with streptomycin.

Most textbooks emphasize the fact that tuberculosis of the middle ear clinically manifests itself as a suppuration. Marx claims that the assumption of older authors that there also exists a dry form of tuberculosis of the middle ear with only an infiltration of the mucosa of the tympanic cavity without exudation has not proven true. My co-worker, U. Russi, however, has seen 25 cases of chronic infiltrative tuberculosis of the lungs, in which the middle ear, behind an intact eardrum, was found to be the seat of a proliferative and infiltrative tuberculosis.

The diagnosis of such a tuberculosis of the middle ear is based on the following: The disease of the ear usually begins with a rapidly increasing deafness combined with a feeling of fullness in the ear. In the beginning there is a striking discrepancy between the rapidly increasing loss of hearing and the slight changes found on the tympanic membrane. The audiogram, at the onset at least, shows that the nerve deafness is more pronounced than the conduction deafness. On the tympanic membrane one first finds a slight inflammation in the region of the manubrium, or a radial injection of the peripheral vessels without retraction of the membrane. Later, the eardrum slowly thickens and the landmarks are obliterated. Yellow foci are often found in the pale red membrane, representing the infiltrations in the submucosa. Along with these alterations in the tympanic membrane hearing grows increasingly worse with a more pronounced conduction deafness. Sometimes it progresses to the point of total deafness. This condition may prevail unchanged for months. Only upon improvement of the basic disease, such as an infiltrative pulmonary tuberculosis, does the proliferative-infiltrative tuberculosis of the middle ear gradually subside. The tympanic membrane, though pale, remains thickened, and recovery of hearing is very rare.

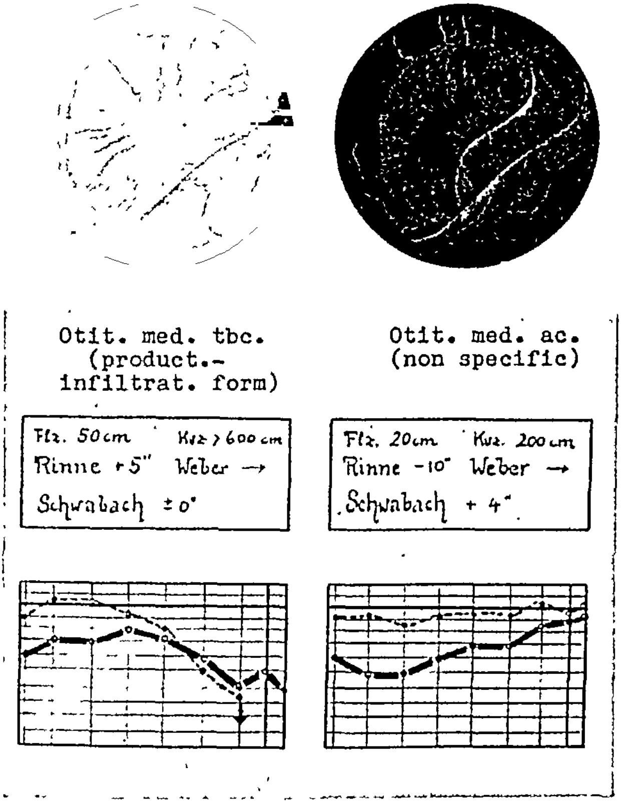


Fig. 3. Proliferative-infiltrative tuberculosis of the middle ear, compared to the characteristic changes and the audiogram in acute otitis media.

U. Russi reports that streptomycin treatment has no noticeable direct effect on the course of a proliferative-infiltrative tuberculosis of the middle ear. At best the antibiotic, by means of its favorable effect on the pulmonary disease, indirectly aids in the healing of the infiltrative tuberculosis of the ear. In our opinion, the specific infiltrations of the middle ear respond so poorly to streptomycin that it helps to prove the tuberculous nature of the infection. In these infiltrations, bacilli are so thinly spread in a tissue supplied by only very few blood vessels that they can hardly be reached by the streptomycin. The very poor tendency toward recovery of hearing, once it has become impaired, also fits into the pic-

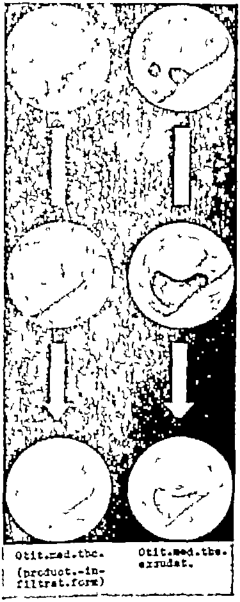


Fig 4 Healing of a proliferative-infiltrative tuberculosis of the middle ear, and improvement of an exudative tuberculosis of the middle ear.

ture of a proliferative tuberculosis of the middle ear; for at best, the specific granulation tissue is replaced by scar tissue which continues to interfere with sound conduction.

Until recently the otologist has been more interested in the toxic effects rather than the therapeutic effects of streptomycin on the ear. The clinical applicability of this new antibiotic seemed questionable. On the other hand, the specific effects of streptomycin on the vestibular labyrinth opened certain possibilities for the exploration of functions of the inner ear. The question of its clinical applicability can now be considered as solved, because we know that loss of hearing as well as disturbances of the vestibular apparatus to a great extent may be avoided by adjusting the dosage of streptomycin to the weight of the patient. For this purpose the daily doses should not exceed 24 mg. of streptomycin per kg. bodyweight, providing the kidneys function normally; furthermore, toxic disturbances of equilibrium and hearing occur even less frequently upon parenteral administration of dihydrostreptomycin instead of streptomycin. C. M. Domon, P. C. Kilbourne and E. Q. King gave a total of 270 gm. of dihydrostreptomycin in daily doses of 3 gm. to an adult without any toxic effects. In our comparative animal experiments the far slighter toxic effects of dihydrostreptomycin have been confirmed.

Van Goidsenkoven and R. Stevens urgently advise against the intrathecal and parenteral application of hydrated streptomycin in tuberculous meningitis. In 173 cases, observed by them over a period of two years, they found decidedly more cases of toxic impairment of hearing after treatment with dihydrostreptomycin than after streptomycin treatment.

The cause of this peculiar sensitivity of the auditory apparatus to the hydrated antibiotic is not known as yet; however, the toxic effects of streptomycin continue to be of theoretical interest even when the therapeutic problems have been solved. The following is a brief description of the functional changes observed in human patients and in animal experiments.

Symptoms from the vestibular apparatus are the first signs of intoxication in the human patient. The vestibular disturbance manifests itself as vertigo occurring when the patient in bed moves his head. This is accompanied by disturbances in vision such as blurring of the surroundings and fogginess. As soon as the patient stops moving his head the dizziness disappears. The disturbance of equilibrium becomes particularly noticeable when the first attempts to walk are made. Following a certain amount of training, the patient is able to walk, at least with his eyes open, but with broad, hesitant steps like a drunken sailor. The surroundings sway before him, and in order to see clearly he must stand still. As soon as he stops walking the dizziness disappears. The Romberg test reveals only a slight swaying in no specific direction. Only occasionally is this dizziness accompanied by a spontaneous nystagmus. Experimental stimulation of the vestibular apparatus through caloric irritation and rotation during the period of dizziness leads to a bilateral decrease and finally to disappearance of the nystagmus. Occurrence of these vestibular disturbances depends upon the amount of streptomycin given. On the other hand, a loss of hearing very seldom occurs even when large doses of streptomycin are given; furthermore, the connection between streptomycin and deafness is not so clear, because in most cases of deafness a tuberculous meningitis is present which might in itself have caused the impairment of hearing. In several of these an improvement of hearing resulted when the streptomycin treatment was stopped, and in some cases, treated with streptomycin, a hearing loss developed in the absence of a tuberculous meningitis.

The special symptoms produced by streptomycin on the statoacusticus are an indication of the seat of the lesions.

A combination of a bilateral disappearance of vestibular irritability with intact hearing, typical for streptomycin intoxication, has already been noted by Guettich in the case of a brain tumor, localized in the floor of the fourth ventricle, as proved by autopsy. The tumor touched the vestibular nuclei. This region was histologically examined by Stevenson and his co-workers in five cases of tuberculous meningitis treated with streptomycin and in three dogs after the application of large doses of streptomycin. In these five clinical cases, and in these three animals, pathological changes were found in the ganglion cells of the nucleus cochlearis ventralis. In addition, two cases showed necrosis, diminution of cells, and hydropic degeneration of the nucleus vestibularis inferior.

For our own streptomycin experiments we used guinea pigs rather than dogs and cats, since the latter animals are both difficult to obtain and usually of various breeds.

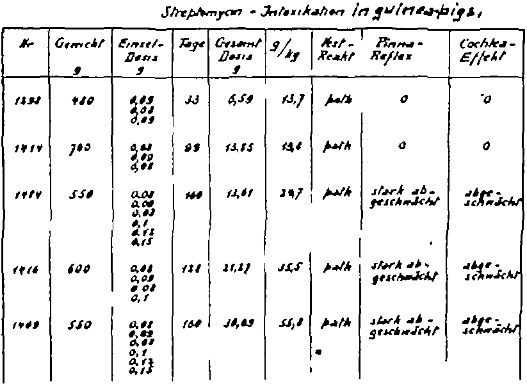
In a first series of guinea pigs treated with streptomycin the histological examination of the animals' petrous bones revealed normal structures in the cochlear and vestibular areas of the internal ear. On examining the serial sections of the guinea pigs' brains we found a decrease in the number of ganglion cells and some glia nodules in the floor of the fourth ventricle in the nucleus triangularis of the VIIIth cranial nerve. We also observed slight changes in the form of plasma vacuolization in the nucleus cochlearis ventralis, and nuclear pycnosis within the ganglion cells.

Shortly afterwards, the late R. Caussé for the first time noticed a loss of sensory hairs in the peripheral vestibular apparatus of three mice poisoned with streptomycin, showing marked disturbance of equilibrium. In additional investigations, Caussé confirmed his first findings of pathological changes within the peripheral vestibular apparatus. The loss of sensory hairs appeared as a secondary process following the degeneration of the sensory cells of the cristae and maculae.

In a second series of experiments, performed on a mouse poisoned with streptomycin, Caussé found degenerative changes in the first cochlear turn of the organ of Corti. The absence of external and internal hair cells was accompanied by a degeneration of the corresponding nervous elements ascending to the ganglion spirale, which showed a decrease in the number of cells. This was followed by K. Berg's report of similar pathological changes in the peripheral auditory organ of streptomycin-poisoned cats which presented distinct disorders of equilibrium.

The cat which had received the highest dose of streptomycin showed no traces of the organ of Corti in the lower and middle cochlear turns. On the same animal the author also observed a certain impairment of hearing.

After Caussé's first communication we reviewed our series of guinea pigs without being able to detect the loss of sensory hairs either on the cristae or on the maculae, which Caussé declared to be characteristic. Assuming that our doses of streptomycin (total doses of 6 to 8 gm) might have been too small to produce visible histological changes in the peripheral organ, we administered to another series of guinea pigs total doses of 6 to 30 gm. of streptomycin in order to control the data reported by Caussé and Berg. Aside from the effects of streptomycin on the vestibular apparatus, we were interested also in a hearing loss due to streptomycin. For the control of sound-perception we first determined during and after the treatment with streptomycin the threshold of Preyer's pinna-reflex for several test sounds which were conducted to both ears simultaneously. Second, we measured on the streptomycin-poisoned animal's right ear the aural microphonic effects immediately preceding vivifixation, as described by Wever-Bray.


Streptomycin - Intoxikation in guineapigs,

Nr	Gewicht g	Einzel-Dosis g	Tage	Gesamt Dosis g	g/kg	Vest.-Reakt	Pinna-Reflex	Cochlea-Effekt
1398	480	0,05 0,08 0,09	33	6,59	13,7	path	0	0
1414	700	0,08 0,09 0,08	98	13,55	19,4	path	0	0
1484	550	0,08 0,06 0,08 0,1 0,12 0,15	140	13,61	24,7	path	stark abgeschwächt	abgeschwächt
1416	600	0,08 0,09 0,08 0,1	128	21,27	35,5	path	stark abgeschwächt	abgeschwächt
1409	550	0,08 0,09 0,08 0,1 0,12 0,15	160	30,69	55,8	path	stark abgeschwächt	abgeschwächt

Fig. 5 Table showing body weight, doses of streptomycin, duration of treatment, results of the equilibrium tests and the sound-perception tests.

For histological examination we finally prepared the petrous bones and the brain of the vivifixed animal, cut them in series, and stained them.

In our experiments with streptomycin five animals gave results which were completely investigated. Table 5 shows body weight, doses of streptomycin administered, duration of treatment, and the results of the equilibrium tests, together with those of the sound-perception tests. All guinea pigs show more or less clearly the typical phenomena of irritation and deficiency of the vestibular apparatus; furthermore, all animals showed a loss of sound-perception to a varying degree during the administration of streptomycin.

The histological examination of the petrous bones reveals changes in the sensory endings within the peripheral vestibular apparatus though varying in extent, similar to those first described by R. Caussé in the ear of the mouse, and afterwards by K. Berg in the ear of the cat. In guinea pigs' ears our findings were: *1.* the cristae (see Figs. 6 and 7) and maculae display a distinct reduction of sensory epithelium.

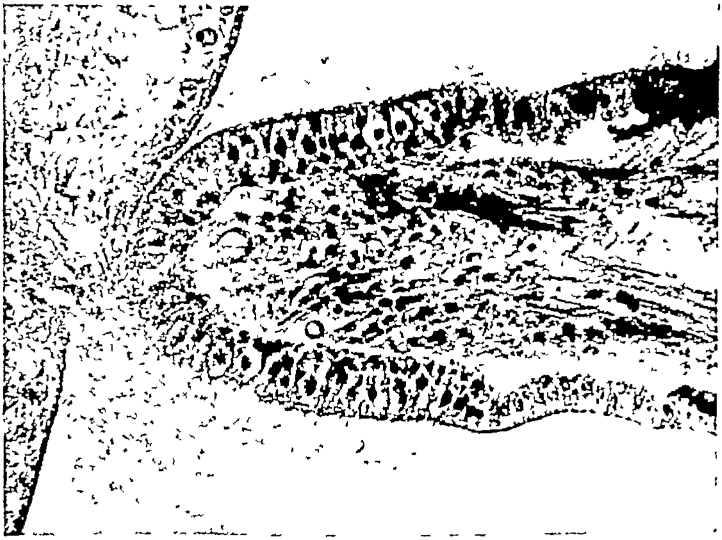


Fig 6 Normal crista ampullaris.

This forms a band in which numerous dark and nearly round nuclei can be recognized but frequently without cellular outlines or cuticular border. The injury starts in the sensory cells, which gradually disappear, while the supporting cells swell and perhaps even multiply, as described by Caussé. Generally the cristae ampullaris and the maculae utriculi show more changes than the maculae sacculi (see Fig. 8).

Fig. 7. Crista ampullaris after streptomycin treatment.

2. The degeneration of sensory cells is accompanied by the loss of sensory hairs, *beginning with the loss of single hairs* and then expanding over a larger surface.

3. Histological examination of the cochlear apparatus of the streptomycin-injured guinea pig's ear reveals more or less pathological changes in all animals, particularly in the organ of Corti, fundamentally similar to the findings of R. Caussé in one mouse, and K. Berg in one cat. In the guinea pig No. 1298 the organ of Corti (see Fig. 9) disappeared symmetrically on either side of the entire area of the first turn. Where

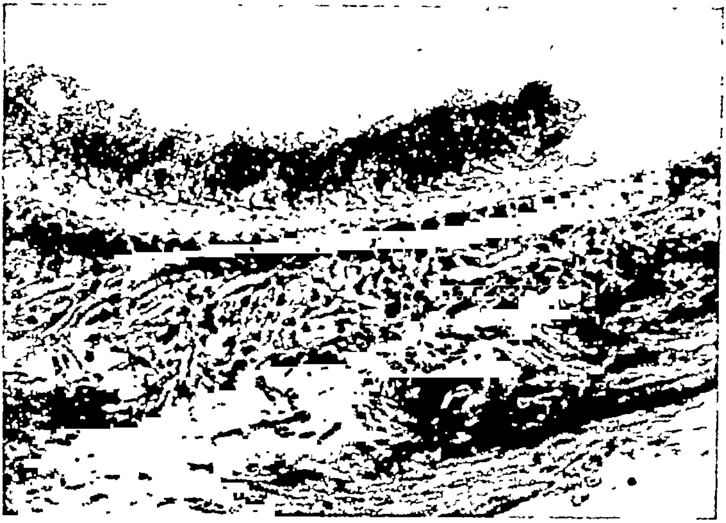


Fig. 8. Macula utriculi after streptomycin treatment.

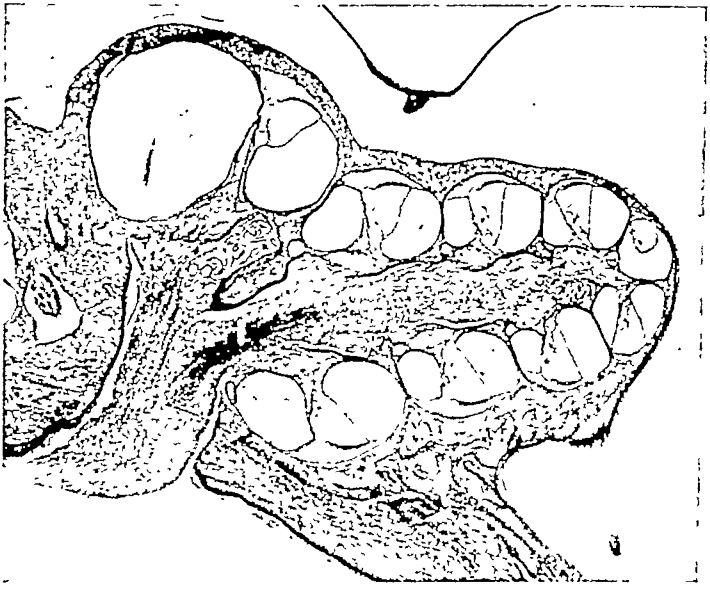


Fig. 9. Cochlea of the guinea pig showing the disappearance of the organ of Corti in the first turn, after treatment with streptomycin.

the organ of Corti has disappeared (see Fig. 10) an ascending degeneration of afferent nerve fibres of the ganglion spirale, changes in the ganglion spirale itself, and further degeneration of the fibres leading to the VIIIth cranial nerve can regularly be demonstrated. The stria vascularis, situated in front of the destroyed organ of Corti also exhibits marked pathological changes.

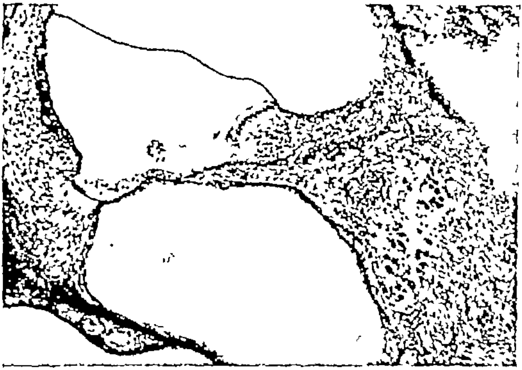


FIG 10 Ascending degeneration of the nerve fibres after treatment with streptomycin.

Histological changes of principally the same kind but of varying degree were found in the inner ear of five guinea pigs poisoned with streptomycin.

The histological examination of the central nervous system of the streptomycin-injured guinea pigs, as in our former findings, shows certain pathological changes in the form of isolated glia nodules and some degeneration of ganglion cells, located in the area of the vestibular nuclei on the bottom of the fourth ventricle and in the nucleus cochlearis ventralis. There is, however no proportion between the extent of these minor pathological signs and the functional loss. Contrary to cases of bilateral Ménière's disease and corresponding bilateral deafness with large doses of streptomycin, giving 4 gm. per day at the rate of 0.5 gm every three hours during three to four weeks. Fowler reached the conclusion that streptomycin is indicated in patients under 50 years of age when customary treatment fails, and there is bilateral deafness. At about the same time C. A. Hamberger, H. Haydén and H. J. Koch also treated four cases of Ménière's disease with large doses of streptomycin. By the daily parenteral administration of 3 gm., a total of 90 gm., two of these cases of bilateral Ménière's disease were kept free from vertigo attacks for a period of five months without any further loss of hearing. Toxic losses in the function of the vestibular apparatus, which manifest themselves around the tenth day of treatment, disappear from one to four months after the end of the streptomycin treatment but continue to be experimentally demonstrable. Two cases of unilateral Ménière's disease also received 3 gm. daily, but a somewhat smaller total dosage, namely, 66 and 55 gm., respectively, was administered parenterally. These patients, too, were free from a Ménière's dizziness at the end of the streptomycin treatment. On the other hand, they suffered severely for months from the functional disturbances of the vestibular apparatus caused by streptomycin. Although the Swedish authors tried to eliminate the affected ear only and made checkups by means of the caloric tests, the sound ear, too, was injured for a long time by the large quantity of streptomycin administered.

Based on these experiences and on the discovery of the peripheral seat of the streptomycin injury, we, too, have treated several cases of Ménière's disease with smaller doses of streptomycin. Three examples of the results up to date are given.

The first case, a 38-year-old man, who for two years has been suffering from bilateral Ménière's disease with marked impairment of hearing in both ears (see Fig. 13) and attacks of dizziness becoming more frequent. The customary conservative methods of treatment mitigated the attacks for only very short intervals. A labyrinth operation at this moment was abstained from on account of the bilateral deafness. A first streptomycin treatment with a total of 24 gm. of streptomycin, given in daily doses of 2 gm., was carried out on this patient weighing 73 kg. On the eleventh day of treatment the patient began to feel slightly unsteady, and treatment was stopped on the following day. The caloric irritability of the vestibular apparatus was somewhat decreased on both sides, the patient felt greatly relieved and remained symptom-free for two months. A new light vertigo attack combined with nausea then occurred, and again we treated him with 2 gm. of streptomycin daily, giving a total of 40 gm. On the thirteenth day of treatment the first disturbances caused by streptomycin began. The patient felt a pressure in his head and a disturbance of his balance upon moving his head. A week later treatment was stopped again. The vestibular apparatus on either side still showed weak responses to caloric stimulation. The toxic disturbances subsided within six weeks. For five months the patient has been completely symptom-free and able to work. The hearing showed neither subjective nor objective changes during the treatment with streptomycin.

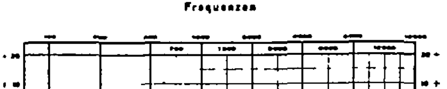


Fig. 12. Thirty-eight-year-old man with bilateral Ménière's disease. Audiogram.

Both in its symptomatology and in its response to streptomycin this case confirms the observations made by Fowler and Hamberger; however, by giving smaller single doses of streptomycin and by keeping the total doses considerably smaller, an equally good therapeutic result was achieved in the course of two treatments without total loss of response to caloric stimulation by the vestibular apparatus.

The second case, a 43-year-old woman with congenital deafness, who had suffered from severe attacks of rotatory vertigo and vomiting for eight years. Various conservative methods of treatment, attempted by otologists, neurologists and neurosurgeons, were unsuccessful. Testing of the vestibular apparatus showed in addition to a very vehement response of the vegetative system, a weak response of the right labyrinth to caloric stimulation, and a spontaneous nystagmus to the left was observed during an attack of dizziness; therefore, a right labyrinthotomy was done, as described by Cawthorne. The attacks of dizziness recurred. A similar operation on the left ear seemed contraindicated, despite the total bilateral deafness, because of the danger of creating a lasting disturbance of the equilibrium in this very sensitive patient. Instead, this woman, weighing 46 kg., received 3 gm. of streptomycin daily for eight days, or a total of 24 gm. During this time the patient vomited severely, and fluid in the form of saline and glucose solution had to be given intravenously. Nine days after streptomycin was stopped, the nausea subsided and the vertigo attacks disappeared. While reading, the patient noticed blurring of type. Three weeks after the end of the streptomycin treatment the patient, completely free from dizziness, was discharged from the clinic. A slight unsteadiness while walking was still present, but that condition, too, soon subsided. We abstained from performing another caloric test on the vestibular apparatus of this extremely sensitive patient. Her condition remained good for one year. Then, in the Summer of 1950, an attack of dizziness with vomiting occurred. Since then these attacks have recurred monthly. The results of the second streptomycin treatment, begun a short time ago, remains to be seen.

This rare case of Ménière's disease in a deafmute patient should serve to demonstrate the situation in which the seat of Ménière's affection could not be located and the patient's condition could not be relieved by labyrinthotomy, but in which by relatively small doses of streptomycin the patient was free for one year from the vertigo attacks which had previously resisted all other therapeutic measures.

The third case is a man, aged 43, with severe vertigo without subjective impairment of hearing. Examination revealed a spontaneous nystagmus in all positions, partly rotatory, partly horizontal-rotatory to the right, becoming more pronounced with shaking of the head. Caloric and rotatory stimulation pointed to a hypersensitivity of the right labyrinth. In the audiogram (see Fig. 14) the depression of the air-conduction curve on either side of 2,000 c.p.s. is more pronounced than usual in a patient of this age. As all other measures failed to cope with the acute attack, 2 gm. of streptomycin daily for four days were administered, making a total of 8 gm. One hour after each injection severe dizziness occurred which subsided the moment the patient lay down. The nausea which constantly annoyed the patient before subsided immediately following the first injection of streptomycin. Two days after the streptomycin treatment was completed the patient, now free from dizziness, left the hospital. On the street he saw people in a certain distance somewhat blurred, and for three weeks he felt very tired. Then, suddenly, he felt perfectly well. He walks and sees normally, and his hearing is intact. No further vertigo attacks occurred for three months. In this case, too, we abstained, for the time being, from a second caloric examination of the vestibular apparatus.

This case represents a first attempt to treat Ménière's disease in its beginning with streptomycin. Since we know today that the antibiotic first affects the peripheral vestibular apparatus, I feel safe in recommending this treatment in cases which are resistant to the customary remedies. That is, providing the doses are kept small and administration of streptomycin is stopped at the right moment.

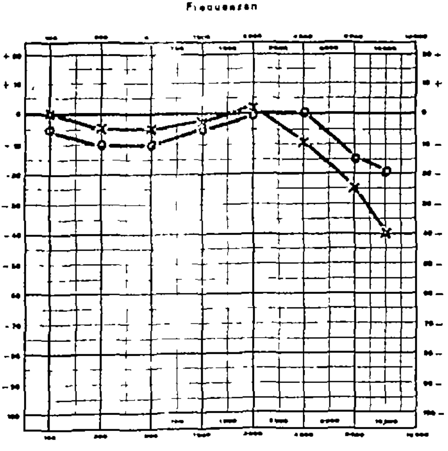


Fig. 14. Forty-three-year old man with Ménière's disease (first attack). Audiogram.

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SUBMUCOUS RESECTION OF THE INFERIOR TURBINAL BONE.*†

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Many local procedures have been advocated in the literature for the relief of perennial nasal obstruction due to enlargement of the inferior turbinate, which cannot be controlled by more conservative general measures. Recent resumés of this subject have been presented by Dixon[1] and Richardson.[2]

Partial or complete inferior turbinectomy has been largely discarded due to the profound physiologic changes such surgery produces; more conservative procedures, however, are beneficial. Such procedures include stripping the turbinal mucosa with a caustic drug or by electrocoagulation. In my experience this has proved to be of temporary value and if carried out too extensively or repeated too frequently results in fibrosis and altered physiology, which may produce symptoms of atrophy.

Intramucosal caustic solutions and electrocoagulation likewise are popular local methods of reducing turbinal enlargement. These procedures produce fibrosis within the cavernous spaces rather than on the surface and are, therefore, preferable to the surface scarifying methods. Care must be taken not to enter too deeply into the soft tissue. Should the cauterizing agent disturb the periosteum of the turbinal bone prolonged discomfort and often sequestration may result.

Submucous resection of the inferior turbinal bone is not a new procedure. Yankhauer, in his student lectures, advocated resection of the inferior turbinal bone, but I have not uncov-

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ered any writings by him on this subject. Harris[3] advocated submucous resection of the middle turbinate in chronic ethmoid disease.

Although this operation has not been popularized in the literature, I find it is being used by a few rhinologists throughout the country. Because it has been a most satisfactory procedure in my experience, I feel justified in making this report.

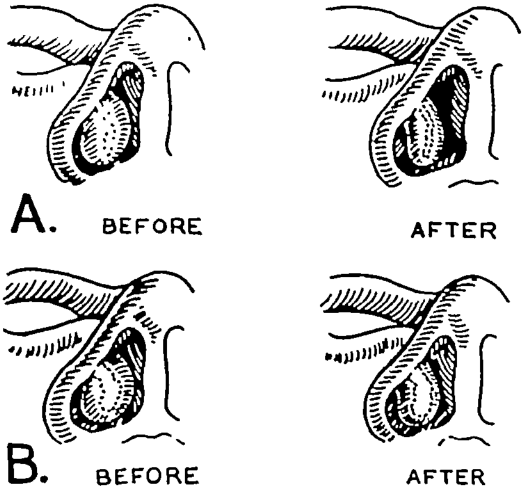


Fig. 1. (A)-Thin turbinal bone. Turbinate shrinks markedly, revealing thin turbinal bone. Intramural cauterization probable procedure of choice in this type. (B) Thick turbinal bone. Turbinate shrinks very little, revealing thick turbinal bone. Submucous resection procedure of choice in this type.

INDICATIONS FOR SURGERY.

Submucous resection of the anterior portion of the inferior turbinal bone is of most value in perennial nasal obstruction due to turbinal enlargement, either unilateral or bilateral, which cannot be satisfactorily controlled by more conservative general management. Included in this group are vasomotor rhinitis, allergic rhinitis, compensatory unilateral hypertrophy secondary to septal deviation, and irreversible

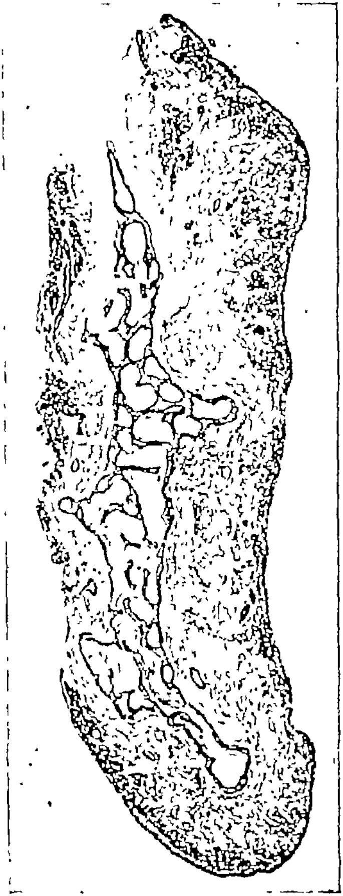


Fig. 2. Cross-section of inferior turbinate reveals rough indented character of turbinal bone surface.

hypertrophy secondary to prolonged irritation. This underlying irritation may be due to infection, occupational environment, excessive smoking, etc.

If the inferior turbinate remains large after thorough shrinkage, it indicates the anterior portion of the turbinal bone is thick. If the turbinate becomes small, it indicates the anterior portion of the turbinal bone is thin. The thicker the turbinate after nasal shrinkage the better the result will be following submucous resection.

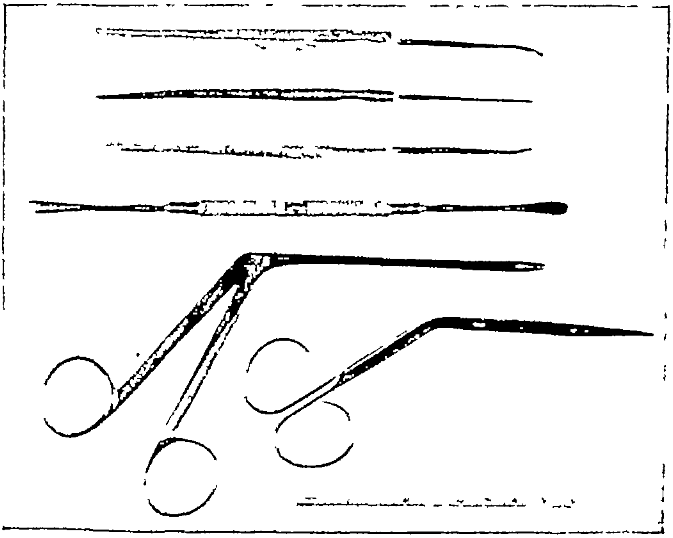


Fig 3 Instruments for submucous resection of inferior turbinal bone. (1) Right and left-angulated and straight Freer knives. (2) Freer elevator used to fracture the inferior turbinate. (3) Takahashi ethmoid forceps used for fracturing and removing the anterior portion of the turbinal bone (4) Small turbinal scissors used for separating the mucosa along the inferior margin of the turbinal bone

If, after nasal shrinkage, the inferior turbinate is very small it indicates a paper-thin anterior turbinal bone with marked soft tissue enlargement. In these cases intramural coagulation, either by caustic solutions or by the bipolar cautery as advocated by Hurd,[4] Owens[5] and others is probably the procedure of choice.

SURGERY.

Surgery of the inferior turbinal bone may be unilateral or bilateral and performed singly or in conjunction with a submucous resection of the nasal septum. We do not advocate this procedure in persons under 16 years of age. The success of this operation depends upon removing the thickened anterior portion of the inferior turbinal bone with a minimum amount of trauma to the surrounding mucosa.

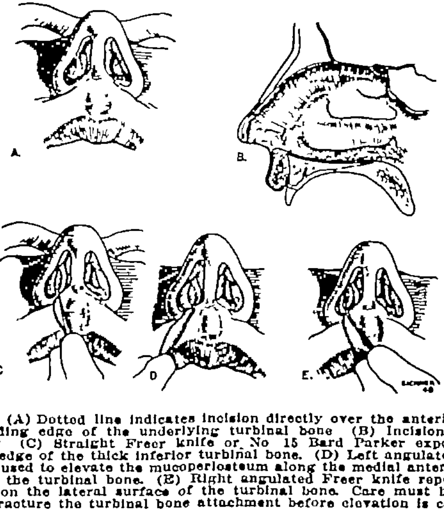


Fig. 1. (A) Dotted line indicates incision directly over the anterior inferior leading edge of the underlying turbinal bone (B) Incision viewed laterally (C) Straight Freer knife or No 15 Bard Parker exposes the leading edge of the thick inferior turbinal bone. (D) Left angulated Freer knife is used to elevate the mucoperiosteum along the medial anterior one-third of the turbinal bone. (E) Right angulated Freer knife repeats the process on the lateral surface of the turbinal bone. Care must be taken not to fracture the turbinal bone attachment before elevation is complete.

The anterior portion of the inferior turbinal bone has a rough and irregular indented surface; therefore, the mucoperiosteum cannot be separated with a blunt elevator, as in septal surgery. A right and left angulated Freer knife, as made by Storz for the purpose, is used to separate the mucoperiosteum by sharp dissection from the lateral and medial surfaces of the anterior portion of the inferior turbinal bone. Tearing of the covering mucosa is thereby avoided.

A small, long-nosed nasal turbinal speculum, as designed by G. O. Proud[6] (the Proud turbinal speculum), is useful as an aid in visualizing the area involved.

TECHNIQUE.

Submucous resection of the anterior portion of the inferior turbinal bone may be performed either as an office or hospital procedure. Premedication and local anesthesia are used as in submucous resection of the nasal septum. Epinephrin-soaked applicators dipped in cocaine flakes are used to anesthetize both the medial and lateral surfaces of the inferior turbinal mucosa. One cubic centimeter of "K" solution (1 per cent novocaine with epinephrin 1:50,000) is injected into the anterior portion of the inferior turbinate for hemostasis and anesthesia.

A straight Freer knife, or No. 15 Bard Parker, is used to make the original incision directly over the anterior inferior leading edge of the turbinal bone.

With the cutting edge of the blade directed inferiorly, the angulated Freer knife is inserted to the previously exposed leading anterior edge of the turbinal bone. By repeated small cutting strokes and with the point of the knife always hugging the roughened turbinal bone, the mucosa is separated from the bone largely by sharp dissection. The mucoperiosteum is elevated for a distance of 1.5 cm. from the anterior leading edge of the turbinal bone. This distance represents the anterior one-third of the turbinal bone and characterizes its thick portion. Care must be taken not to apply excessive pressure on the turbinal bone during this elevation. If fracture occurs before the mucoperiosteum is separated from the medial and lateral surfaces, further separation is most difficult due to the mobility of the underlying fractured bone. For this reason the lateral side of the turbinate is separated first, and if fracture should inadvertently occur, the medial surface is more readily visible, and the lateral wall in the nose can be used as a support while the mucoperiosteum separation is completed from the medial aspect of the turbinal bone.

After separation of the mucoperiosteum from both the medial and lateral surfaces of the bone has been completed for a distance of 1.5 cm., or approximately one-third the length of the inferior turbinal bone, the attachment of the mucoperiosteum is severed from the inferior edge of the inferior turbinate for an equivalent distance with the small turbinal scissors. The anterior thick portion of the inferior turbinal bone, which is now separated from the mucoperiosteum in its entirety, is grasped with the Takahashi forceps, rotated, fractured, and gently removed.

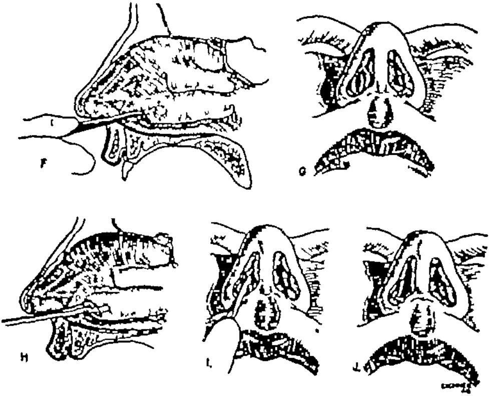


Fig. 5 (*F*) *Illustrates* motion of Freer knives. (G) Completely separated mucoperiosteum. Scissors are used to complete this process along inferior margin of turbinal bone (H) Takahashi forceps is ideal for grasping and removing anterior one-third of the turbinal bone (*I*) Elevator is used to fracture remainder of turbinate laterally (*J*) Now position of thinned inferior turbinate. Note increased nasal airway.

If there is a tendency for the bone to cling to the mucoperiosteum, additional exploration must be done to determine the area of continued attachment. This attachment is then severed completely with the scissors. Forcible removal of the bone in this instance will tear the mucosa and result in altered physiologic surface changes which we are striving to avoid.

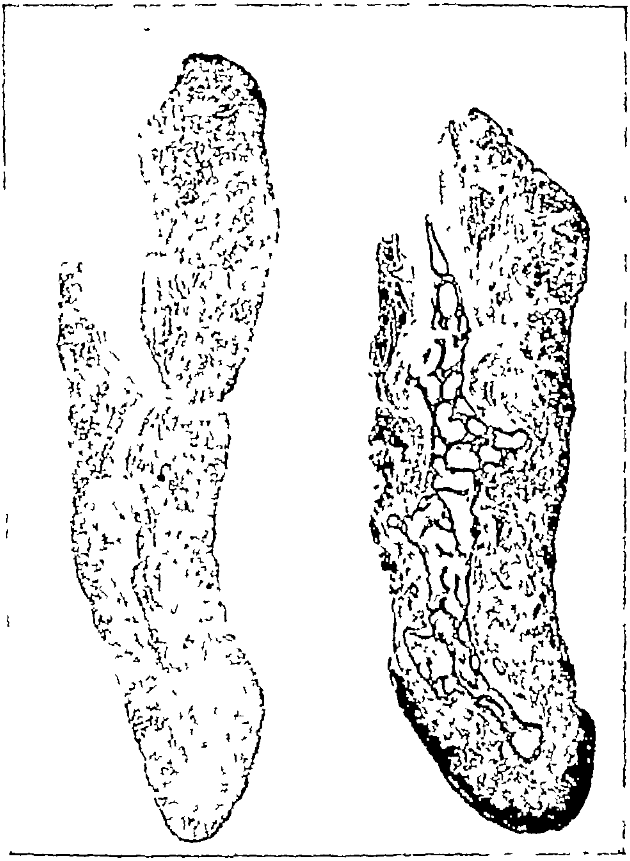
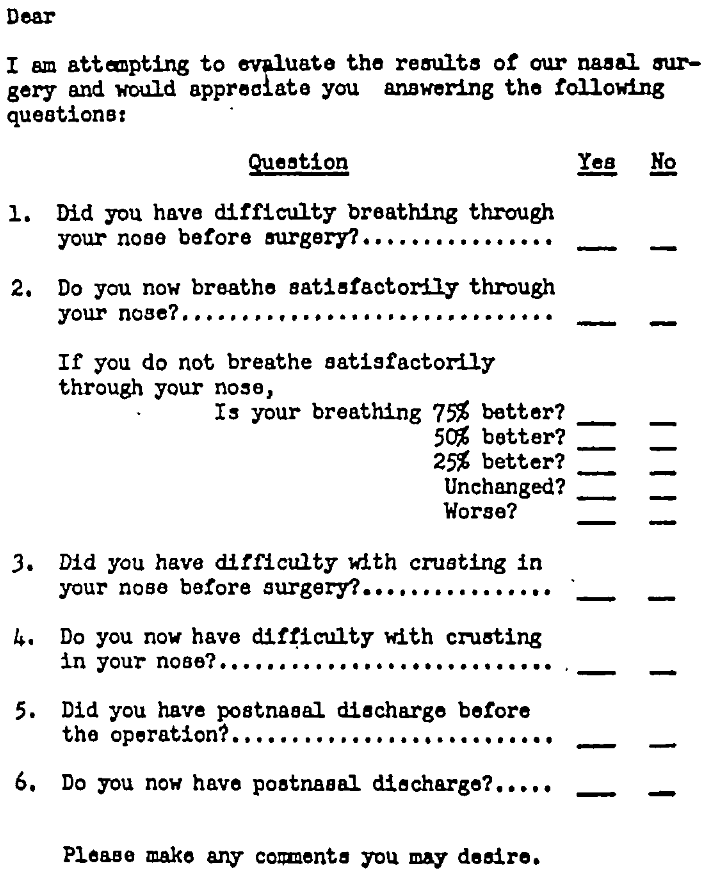


Fig 6 Cross-section of inferior turbinate before and after removal of inferior turbinal bone

The remaining two-thirds of the turbinal bone is tissue paper-thin and need not be removed. A Freer elevator is used to infract the remaining bone, first medially and then laterally. This immediately opens the nasal airway and if the posterior tip of the turbinate is bulbous or papillomatous in character, it can be cauterized or removed with a small snare, as the operator may choose.

Of this total, 23 cases have been discarded due to incomplete follow-up examinations and questionnaires.



Dear

I am attempting to evaluate the results of our nasal surgery and would appreciate you answering the following questions:

Question	Yes	No
1. Did you have difficulty breathing through your nose before surgery?................	___	___
2. Do you now breathe satisfactorily through your nose?...............................	___	___
If you do not breathe satisfactorily through your nose, Is your breathing 75% better?	___	___
50% better?	___	___
25% better?	___	___
Unchanged?	___	___
Worse?	___	___
3. Did you have difficulty with crusting in your nose before surgery?................	___	___
4. Do you now have difficulty with crusting in your nose?...........................	___	___
5. Did you have postnasal discharge before the operation?..........................	___	___
6. Do you now have postnasal discharge?.....	___	___

Please make any comments you may desire.

Fig. 7. Results were tabulated both by the above questionnaire and postoperative observation.

SUMMARY.

Irrespective of valiant attempts to control cases of turbinal enlargement by general therapy, a certain percentage of cases have proven unmanageable in my experience. Various local methods of surface and intramural scarification have been used in an attempt to improve the nasal airway of these individuals.

	Preop.	Postop.
Crusting	48 or 47%	52 or 51%
Postnasal discharge	65 or 63%	58 or 57%

Fig 8 There was no significant change in the degree of crusting before and after surgery. This would indicate little tendency toward atrophic changes. Only 6 per cent of the cases revealed a significant decrease in the amount of postnasal secretion This would indicate increased ventilation does not necessarily aid in diminishing postnasal discharge.

Submucous resection of the anterior one-third of the inferior turbinal bone has given our best results in selected cases. This is especially true if the inferior turbinate remains relatively thick after thorough shrinkage, thereby indicating a thickened underlying turbinal bone in its anterior portion.

Complete relief of obstruction	47 or 46%
75% relief of obstruction	19 or 18%
50% relief of obstruction	14 or 14%
25% relief of obstruction	5 or 5%
Unchanged	14 or 14%
Worse	3 or 3%
	102 or 100%

Fig 9 The above figures indicate the majority of patients were either totally relieved or approximately 75 per cent improved two years or more after having had a submucous resection of the inferior turbinate performed.

CONCLUSION.

1. Submucous resection of the anterior one-third of the inferior turbinal bone is recommended for uncontrollable perennial inferior turbinal enlargement.

2. The procedure is herein described in detail together with certain instruments developed specifically for the operation.

3. A study of 102 cases over a two-year period indicates a favorable response to this surgery.

4. This operation reduces the size of the inferior turbinate with a minimum of interference to the physiology of the turbinal tissue.

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RAPID ANTIBIOTIC SENSITIVITY DETERMINATIONS FOR PSEUDOMONAS AERUGINOSA ISOLATED FROM EXTERNAL OTITIS.*

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INTRODUCTION.

One of the major problems encountered in the therapy of otitis externa infections is the lack of information concerning the etiologic agent involved and the sensitivity of the causative agent to the available antibiotics or other therapeutic agents. Several workers[1,3,7] have reported that a large proportion of the cases of otitis externa are infected by *Pseudomonas aeruginosa (Bacillus pyocyaneus).*

This paper reports procedures that reveal in a relatively short time the presence of *P. aeruginosa* and its reactions to a battery of several antibiotics and to a drug, sulfamylon. Sulfamylon (p-aminomethylbenzene-sulfonamide) has been shown to be a very effective treatment for infections with *P. aeruginosa* as the etiologic agent.[2,4,5,6]

MATERIALS AND METHODS.

The studies were performed on 54 cultures of *P. aeruginosa* obtained from frank cases of otitis externa.† Each isolation was made from a specimen collected on a sterile cotton-tipped swab and placed in a test tube containing brain-heart infusion broth. This was brought immediately to the laboratory, where the required culture procedures were done. Two media were

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†The patients were seen in the Department of Otorhinolaryngology, School of Aviation Medicine

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employed: Proteose No. 3 blood agar, because it promotes excellent growth of microorganisms usually found in the ear; and an asparagin agar because the blue-green pigmented colonies of *P. aeruginosa* were easily detected on this medium within the period of incubation employed. Separate plates of blood agar and asparagin agar were inoculated with each of the contaminated swabs. The plates were streaked with an inoculating loop so as to produce a surface covered with isolated colonies. At the end of 18 to 24 hours' incubation at 37° C. the plates were examined for colonies characteristic of *Pseudomonas* species. Such colonies were picked to slants of Kligler's iron agar medium and incubated overnight to determine their reactions. Those cultures with the usual appearance of *Pseudomonas* were subjected to certain of the criteria listed for this genus.[8] Cultures identified as *P. aeruginosa* were employed in this study.

The brain-heart infusion broth, Kligler's iron agar, and proteose No. 3 agar (to which 5 per cent defibrinated rabbit blood was added) were made from dehydrated culture media.* The asparagin agar was formulated in this laboratory, as follows:

Asparagin	3.0	gm.
Bacto-tryptose	5.0	gm.
Dextrose	2.0	gm.
Dipotassium phosphate	2.0	gm.
Magnesium sulfate	1.0	gm.
Potassium nitrate	0.5	gm.
Bacto-agar	20.0	gm.
Distilled water	1,000	ml.

Heat to boiling to dissolve the agar, adjust the pH to 7.2 and sterilize in the autoclave 15 minutes at 15 pounds pressure. Cool to 47° C. before pouring plates.

The sensitivity of these *Pseudomonas* organisms to several antibiotics and to sulfamylon was determined by the following method: The surface of a culture plate (asparagin agar) was inoculated with an 18- to 20-hour culture of the test organism. With a sterile, cotton-tipped swab a drop of the culture (1:1,000 dilution) was spread over the entire surface of the medium. Filter paper disks impregnated with various concentrations of each of the antibiotics and of the sulfamylon were then placed on the culture. A general method for the use of these disks has been *described.*[9]

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*Bacto-Difco Laboratories, Detroit, Mich.

The three different concentrations used for each of the antibiotics and the sulfamylon were labeled A, B and C (see Table 1).

TABLE 1.

LIST OF THERAPEUTIC AGENTS USED IN DETERMINING THE SENSITIVITY REACTIONS OF PSEUDOMONAS AERUGINOSA.

Test Agent	Concentration A	Concentration B	Concentration C
Sulfamylon*	15 mcg.	40 mcg.	160 mcg.
Chloromycetin†	10 mcg.	30 mcg.	60 mcg.
Terramycin	10 mcg.	30 mcg.	60 mcg.
Dihydrostreptomycin	0.5 mcg.	1.0 mcg.	10 mcg.
Streptomycin	0.5 mcg.	1.0 mcg.	10 mcg.
Penicillin	0.5 unit	1.0 unit	10 units
Bacitracin	2.0 units	10.0 units	20 units

*The sulfamylon hydrochloride powder was supplied by the Medical Research Department of Winthrop-Stearns, Inc.

†The antibiotic disks are now available from Difco Laboratories.

The concentrations employed were such that if zones of inhibition were produced around disks A, B and C for a given agent the test organism was sensitive. Inhibition zones around disks B and C and not around A indicated slight or questionable sensitivity. A resistant organism was indicated when no inhibition occurred or when inhibition was produced around only the C disk.

COMPARATIVE SENSITIVITY TESTS.

Each of three plates of asparagin agar was inoculated with an 18- to 20-hour broth culture of the *Pseudomonas* organism. A sterile, cotton-tipped swab was used to spread a drop of the culture (1:1,000 dilution) over the medium.

When the inoculation was completed the antibiotic and sulfamylon disks were placed on the surface of the medium in a definite arrangement. The disks were so spaced that a zone of inhibition could be easily noted and measured. The pattern permitted testing of all seven agents within a group concentration on a single plate (see Fig. 1).

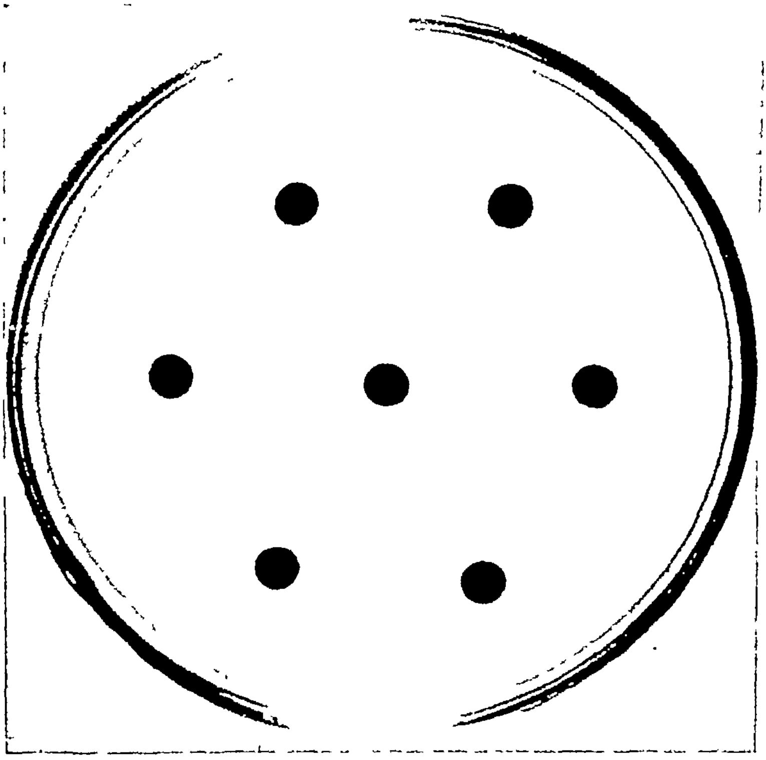

Fig. 1. Pattern used for spacing of antibiotic and sulfamylon disks on the culture media plates inoculated with either pseudomonas aeruginosa or material from an infected ear. Photograph by: U. S. A. F. School of Aviation Medicine.

The use of this pattern required three plates, one for each of the three concentrations A, B and C (see Table 1) to make a comparison of all agents under study. The inoculated plate was placed over the pattern, and with flamed forceps the disks were removed from their containers and placed on the inoculated surface according to the pattern (see Fig. 1). This was done for concentration groups A, B and C for all isolations of *Pseudomonas* under study. The plates were examined after incubation for 18 to 24 hours at 37° C. The zones of inhibition were easily defined as clear zones surrounded by growth of blue-green pigmented colonies on the asparagin medium and as nonhemolyzed zones surrounded by partially hemolyzed areas when blood agar medium was used. The appearance of the test plates at the end of 18 hours' incubation indicated that sensitivity could be determined in even less time. Culture plates with *P. aeruginosa* growth and showing the typical zones of inhibition present after 18 hours' incubation are presented in Figs. 2, 3 and 4.

Studies were made to determine the time required before an adequate sensitivity reading could be made. This was done

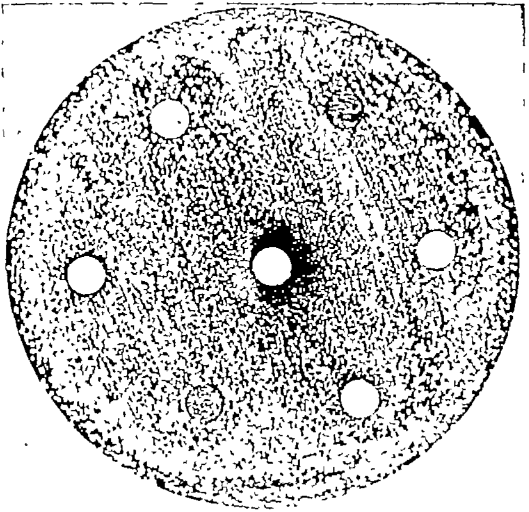

Fig 2 Inhibition zone produced around the sulfamylon disk only. Pseudomonas aeruginosa sensitive only to sulfamylon. Photograph by: U. S. A. F. School of Aviation Medicine.

microorganisms is now used routinely for all specimens submitted to this laboratory for bacteriological analyses. A preliminary or tentative report as to antibiotic sensitivity and identity of the etiologic agent can now be made to the physician much sooner than before.

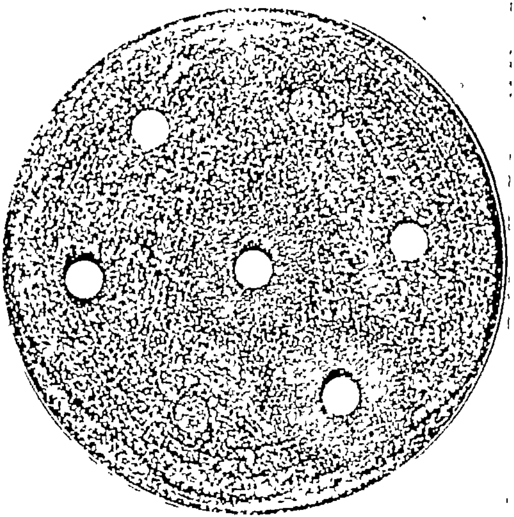

Fig. 4. Inhibition zones produced around the sulfamylon and chloromycetin disks. Pseudomonas aeruginosa culture sensitive to both sulfamylon and chloromycetin. Photograph by: U. S. A. F. School of Aviation Medicine.

RESULTS.

Cultures of *P. aeruginosa* were tested for sensitivity to three different concentrations of each of seven different agents. The results are presented in Table 2.

All of the 54 isolations of *P. aeruginosa* studied were susceptible to either sulfamylon and/or chloromycetin. Among the 52 isolations found sensitive to sulfamylon, six reacted

TABLE 2.

RELATIVE SENSITIVITY OF PSEUDOMONAS AERUGINOSA TO DIFFERENT THERAPEUTIC AGENTS.

Test Agent	Concentration Groups in Which Inhibition Zones Were Produced A	 B	 C
Sulfamylon	52	54	54
Chloromycetin	45	54	54
Terramycin	19	54	54
Dihydrostreptomycin	2	2	48
Streptomycin	0	2	47
Penicillin	0	0	0
Bacitracin	0	0	0

only to this agent and to none of the others tested. These six cultures were isolated from four different patients. Only one culture reacted to chloromycetin alone. Terramycin was the only additional antibiotic to indicate usefulness as an agent active against this organism. Dihydrostreptomycin and streptomycin showed a poor degree of activity in comparison with that shown by sulfamylon, chloromycetin, and terramycin. Penicillin and bacitracin were not effective in the concentrations employed. *P. aeruginosa* cultures tested with aureomycin sensitivity disks showed no reaction or, at the most, only a slight reaction. Aureomycin, therefore, proved even less effective than streptomycin.

DISCUSSION.

This method of determining the sensitivity of *P. aeruginosa* to various therapeutic agents can more quickly aid the physician in his choice of therapy. The determination was even more rapid when the disks were used on the primary media plates inoculated with infected material taken directly from the patient. When *P. aeruginosa* was the etiologic agent involved, a preliminary report as to the effectiveness of certain antibiotics could usually be made to the physician within eight to 10 hours after fresh culture material was submitted to the laboratory.

SUMMARY.

Determinations were made of the sulfamylon (p-aminomethylbenzene-sulfonamide) and antibiotic sensitivities of 54 cultures of *P. aeruginosa* isolated from cases of otitis externa. Filter paper disks impregnated with suitable concentrations of sulfamylon and the antibiotics were placed on the surface of agar plates inoculated with *P. aeruginosa.* These plates were incubated at 37° C. and examined at intervals for growth inhibition around the various disks Stop-motion photography of these plates revealed that sufficient growth of *P aeruginosa* occurred within 6 to 8 hours to indicate well defined zones of growth inhibition around those disks to which the organism was sensitive. A procedure is described for the rapid determination of the sulfamylon and antibiotic sensitivity of *P. aeruginosa* and other microorganisms present in a specimen submitted to the laboratory for bacteriological analysis.

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SOCIALIZED MEDICINE.*

JOHN W. CLINE, M.D. (by invitation),
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When the invitation was issued to discuss the subject of socialized medicine, I was a bit disturbed because it is a tremendous subject. It seemed, however, that there was no need to go into details concerning its disadvantages from the medical standpoint. You are aware of what has occurred on the Continent, in England and New Zealand under programs of socialized medicine. There has been some confusion concerning what actually constitutes socialized medicine. It is my opinion that any program of medical care for a large portion of our population which is tax supported and governmentally controlled is socialized medicine. The advocates of such programs, from President Truman on down, have made a strenuous effort to avoid the term. Restricted and artificial definitions and adroit terminology, however, have provided them no escape. A recently elected United States Senator stated that there was no magic in words and that any governmental program for medical care was socialized medicine regardless of what it is called.

American medicine faced a great threat to its freedom as the result of President Truman's re-election in November, 1948. He had disregarded the pronouncement of his party and campaigned on a personal platform, promising socialized medicine among many other items. The advocates of such schemes and many of the commentators and columnists believed that his re-election was a "mandate" to enact such legislation.

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*Read at the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., San Francisco, Calif., Jan. 27, 1951.

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The American Medical Association House of Delegates met in St. Louis about one month after the election. It issued a ringing statement of principles, directed a campaign against socialized medicine and levied a $25.00 assessment upon the membership to finance that campaign. The campaign was begun in the Spring of 1949, and the situation less than two years later has undegone tremendous change.

The campaign was designed to convince the people of this country of the harm to their medical care which would result from socialized medicine, to show them the disadvantages of loss of freedom in securing medical care and the hazard to other aspects of their freedom involved in the prospect of wider socialization. At the same time, an effort was made to educate the public to the advantages of voluntary health insurance as a mechanism of spreading the cost of illness.

The response of the medical profession throughout the country was encouraging, although not all that was hoped for. Most of the state and county medical societies participated vigorously and effectively in the campaign, but far too many individual physicians did not. Many of our outstanding physicians, who would be adversely affected by any program of socialized medicine, seemed apathetic to the campaign. This was a problem at the outset and still remains a major problem.

There is considerable overlapping between the groups who attend purely scientific meetings and those who take an interest in the affairs of medical organization. On the other hand, many who are prominent in scientific activities lack interest in protection of the freedom essential to maintenance of high scientific standards. There is far too much of an attitude of letting the other fellow do it.

The preservation of freedom in medicine, as preservation of freedom in general, is the job of everyone. Unless there is much wider participation in this effort, the hazard will not be permanently overcome.

There are certain left wing groups and others who actually favor socialized medicine. Some of these have undergone a change of heart but some still remain openly antagonistic to the campaign of the Association. Probably nothing can be done to change their minds, and it probably would be a waste of time to try. They are numerically unimportant, even though some of them are quite vocal. Some of them have become less vocal as the cause has lost popularity.

The presence of such people in the profession makes it even more essential that we have an otherwise unified, militant and vigorous profession.

The results of the present campaign can be seen in the change in the character of news stories and the articles in our various periodicals. Formerly, the great bulk were opposed to our point of view, but now the majority are favorable. .Over 90 per cent of the newspapers of the country are editorially on record against socialized medicine. About 11,000 organizations of national, state and local scope have passed resolutions against compulsory health insurance. Certain public opinion polls show very favorable trends away from the belief that socialized medicine is either necessary or desirable.

The active controversy between the socializers and the American Medical Association has stimulated great interest in matters of medical care. The growth of voluntary health insurance has been tremendous. In fact, it is still so rapid that by the time the figures are compiled, they are already out of date. At the present time about 20,000,000 people have Blue Shield or other medically sponsored coverage, 40,000,000 are enrolled in Blue Cross, and over 72,000,000 have coverage of some type against the cost of illness. By the end of this year, the number should be more than 80,000,000, and I believe we can confidently look forward to the enrollment of the vast majority of our population within the next few years.

The controversy also has established the hazard of creeping socialism in the minds of many of our people. More and more people see the threat to their own future freedom of activity and endeavor which the socialization of any segment of private enterprise carries with it.

The one large segment of our population which we have not been able to reach, at least as far as its higher echelons are concerned, is organized labor. Even here, there are some encouraging signs.

In December, Mr. William L. Hutcheson, President of the International Carpenters Union, which has 700,000 members, presented a paper entitled, "Socialized Medicine Is No Bargain," to the American Medical Association meeting in Cleveland. Some of you may have seen this paper and it will be distributed ultimately to all the physicians in the country. Within the next year we should see rather marked change on the part of certain other labor leaders.

I have talked with some who are not in the highest positions but yet are of importance who recognize clearly that socialization of medicine would ultimately destroy the freedom of labor. This point of view seems to be growing. It is, therefore, important for physicians to utilize the opportunity to talk to their patients who are members of labor unions, and particularly officers of labor unions, and to emphasize this danger to them. If enough labor union members and officials become convinced of the threat to labor, the official attitude of labor will change and the one large segment of the population which has been vigorously opposing us will cease its activity in behalf of socialized medicine.

The recent elections indicate a strong trend away from socialized medicine. In the larger states, it is difficult to interpret the effect of single issues. The importance of the political activity of physicians as individuals and in informal groups has been apparent in certain close elections and in the elections in certain of the smaller states and in congressional districts. Very few candidates who openly espoused socialized medicine survived the last election. When a candidate espouses socialized medicine, he usually advocates other causes of similar orientation; therefore, the last election probably indicated a trend away from socialistic proposals in general.

Certain important candidates felt it advisable to disavow socialized medicine completely. Some made the effort to escape by claiming that the administration's plan was not socialized medicine, but others repudiated compulsory health insurance by name.

Senator Pepper, of Florida, in a radio interview attributed his defeat to the activity of the medical profession during the primary election. I have been informed that Senator Graham, of North Carolina, and Senator Taylor, of Idaho, likewise attributed their primary defeats to the activities of the medical profession. Senator Thomas, of Utah, was the author of the administration's omnibus bill for socialized medicine in the 81st Congress. He has attributed his defeat to that fact.

In Milwaukee, Andrew Biemiller, who was the most vigorous and vicious assailant of American medicine in Congress, and had announced that he was the spokesman for the administration's measure, was beaten largely on the issue of socialized medicine. The physicians in Milwaukee did a tremendous job in accomplishing his defeat. The same can be said of Mr. Sullivan, of Omaha.

One of the other lessons to be learned from the last election comes from Ohio. Senator Taft had the full force of the administration, the American Federation of Labor, the Congress of Industrial Organizations, the United Mine Workers and the Railroad Brotherhoods arrayed against him. Their campaign was particularly violent and used comic strips and scurrilous material to discredit Mr. Taft. He carried 84 out of 88 counties, and every industrial area in Ohio.

This clearly showed that the American working man is an intelligent individual who will make up his own mind when the facts are presented to him. Certainly, Mr. Taft did not attain majorities in the industrial areas of Ohio because he received the votes of business men and employers. It was the laboring people of Ohio who demonstrated that they could not be led into blindly following the exhortations of the labor union officials. Neither labor nor any other group can be so influenced.

When the individual citizen is alone with his conscience in the polling booth, he votes upon the basis of his convictions and not according to the dictates of his leaders. This, I believe, emphasizes the need for greater activity on the part of the profession in general to convince the individual citizen of the dangers of socialized medicine.

As the result of the crystallization of public sentiment against socialized medicine, the administration supporters in the 81st Congress recognized that no legislation of this type could be passed during that Congress. They, therefore, decided to center their attack upon the so-called "fringe bills." The two major ones of these were Federal Aid to Medical Education and Federal Aid to Public Health Departments.

The attitude of the American Medical Association with reference to both of these proposals has been grossly misinterpreted. Our opponents have said that we have resorted to restrictive measures to limit the number of physicians in this country and that we are opposed to Public Health. You, of course, know that nothing could be further from the truth.

The country could use some additional physicians, and we would like to see the number increased, but only if the current high standards of medical education are continued. To lower them would be ruinous to the future of medical care.

There are only a few methods of increasing the number of physicians. We could resort to an accelerated program such as that which was in effect during World War II, but most of us who had experience teaching under that program hope never to see it reinstituted. The faculties became fatigued and did not do as good a job of teaching as they should have done, *but more important is that the progressive fatigue of* the students made it difficult for them to absorb what was presented to them. I observed this with my own students and feel that we should not resort to such a program again.

We can build new medical schools, but this takes a great deal of time. In one instance, the appropriations for a new school were made almost 10 years ago, and as yet no students have been enrolled. Where concentration of population makes medical schools feasible and they can be adequately supported, they will develop in the course of time.

Some of the two-year schools could expand their curricula to complete the course of medical education, but some are in areas where clinical material would not be available. The medical curriculum could be shortened, as was done in Germany during the war, but this again would reduce the quality of medical education.

The enrollments of our medical schools have been materially increased, but there is a limit to the increases which may be undertaken without sacrifice of quality of education. The individual school must make the decision as to how great an expansion it may undertake without destroying its teaching program.

Some of our medical schools are in financial distress. The American Medical Association recognized this fact and voted a half million dollars to stimulate realistic private support for our medical schools. It was necessary to establish a foundation to receive these funds and money which has been contributed by other organizations and individuals. This fund will be merged in the future with a fund for medical education which is now in the process of formation but is not ready to be announced.

It is our belief that a large enough sum can be raised to augment the present income of the medical schools to the point that their difficulties in carrying out their teaching programs can be relieved. In addition, it is likely that the American Medical Association will support one-time federal grants for remodeling and construction under a program similar to the Hill-Burton Bill for the construction of hospitals.

In this way, need for federal support for continuing operation of our medical schools can be avoided. There is great danger of federal control if subsidies for operation are permitted.

Subsidies bear a certain resemblance to narcotic drugs. They produce an illusory sense of well-being, they make addicts of the recipients and the withdrawal symptoms are horrible. The mere threat of withdrawal of funds would compel compliance to the wishes of the bureaucrats in Washington in most instances. Even if those responsible for running our medical schools were of sufficiently rugged character to refuse to comply with the wishes of the bureaucrats, they would face another source of pressure.

In 1942, in reference to one of the farm programs, the Supreme Court handed down a decision in which Mr. Justice Jackson said, "It is not lack of due process that the government may regulate that which it subsidizes."

While this decision did not concern education or medical education, the principle of law that the Federal Government may regulate that which it subsidizes is clearly established and stands unreversed. The ominous nature of this decision is readily apparent.

We must, therefore, continue to oppose subsidization of medical education and rely upon private support upon a wide base. I hope that every physician in the country will contribute generously and promptly to aid our medical schools.

The situation with reference to subsidization of local public health units is somewhat similar. As the legislation has been drafted, it would place dictatorial powers in the hands of the United States Public Health Service and permit it to direct local public health activities. We believe that the autonomy of local public health officials should be preserved.

The American Medical Association has advocated a Department of Health, headed by a physician with Cabinet status, since 1873. The Association and its members were mainly responsible for the development of public health activities in the United States. The American Medical Association has continuously supported the public health movement ever since.

It is our opinion that public health is primarily a local problem, secondarily a state problem, and that the Federal Government should interest itself in local public health matters only when the local unit of government and the state government have fallen down. We further believe that any subsidy to local public health activities should be to educate the community which does not have such a program to its value. Subsidies should be for limited periods of time with the expectation that the local unit of government would then support its own public health program. If it did not see fit to do so at the end of a brief period of years, federal support should be withdrawn.

At the present time, we face a grave international situation. There will be many proposals for medical care under the guise of wartime emergency. Some of these may be legitimate projects, but there is great danger that some variety of socialization of medicine will be introduced by executive action or as a portion of some bill which otherwise is a legitimate measure in preparation for war.

We must be continuously alert to see that the present emergency is not perverted into legislation or executive action which will accomplish socialized medicine by oblique methods. One such proposal was included in the War Man Power Act but was eliminated after the opposition of American Medicine had been expressed.

We must not permit the fallacious arguments of alleged lack of patriotism or hindering the war effort to prevail and destroy our liberty while we are presumably engaged in defending it.

On the subject of socialized medicine, the 82nd Congress is more sympathetic to our point of view than was the 81st, and temporarily there is no hazard of direct, honest legislation to accomplish compulsory health insurance. This situation, however, may only be temporary if we do not maintain the activity to prevent it.

We have come a long way in the past two years, but this advance will go for naught if we do not remain vigilant and vigorous. We cannot relax in our effort to convince the people of the hazards of socialized medicine and of socialism in general. We cannot relax in our political activities as individuals and in informal groups until these issues have been thoroughly resolved.

We know the great disadvantages of socialized medicine, and we know the harm of socialism in general; also, one cannot envision an island of medical freedom as standing long in a sea of socialism.

We have a great responsibility to the American people, to ourselves and to our successors to see that these things do not come to pass.

AMERICAN OTOLOGICAL SOCIETY, INC.,

AWARD OF MERIT.

For their joint research contributing to the knowledge of "The Embryology in Anatomy of the Temporal Bone and the Ear" medals were awarded to Barry G. Anson, Ph.D., Professor of Anatomy, Northwestern School of Medicine, Chicago, Ill., and Theodore Bast, Ph.D., Professor of Anatomy, Wisconsin School of Medicine, Madison, Wis.

The Award of Merit medal is accompanied by an honorarium and is granted by the American Otological Society, Inc., for the purpose of recognizing outstanding service in research in otology and in the interest of encouraging research in the problems of impaired hearing.

TRACHEOTOMY IN BULBAR POLIOMYELITIS.*

GEORGE O. CUMMINGS, JR., M.D.,
Syracuse, N. Y.

In recent years there has been an increased incidence of poliomyelitis. Adults have been afflicted in greater relative numbers than in the past, and there have been more bulbar cases reported than ever before. Tracheotomy has been used with excellent results. Although Wilson[1] first used tracheotomy for bulbar poliomyelitis in 1932, it was largely the reports of Galloway[2] and Priest, Boies and Goltz[3] that made tracheotomy become a well recognized and in many places an accepted procedure. A review of the literature failed to reveal a paper which correlated the physiopathology of the disease to the clinical course and therapy and discussed the mechanical armamentarium available.

In the Summer and Fall of 1950, in Syracuse, N. Y., there were 334 cases of poliomyelitis. Sixty-eight were bulbar. Twenty cases were severe with pharyngeal obstruction. Six tracheotomies were done. A 73-bed communicable disease hospital was used during the epidemic for the treatment of these cases. Every bulbar or suspected bulbar case received an ear, nose and throat consultation on admission. This was a precedent established that year by the combined attending staff.

In order that the laryngological consultant may be of more value to the patient and to the other services, he should be cognizant of the neuroanatomy and neurophysiology of the disease. The nuclei of the IXth and Xth cranial nerves which control the act of deglutition lie in the region of the medulla and adjacent fourth ventricle. They are the dorsal nucleus of the vagus and the nucleus ambiguous with the tractus soli-

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tarius intervening. The afferent nerve fibres from the pharyngeal plexus run to these nuclei. The circulatory center lies in or near the dorsal nucleus of the vagus, and the respiratory centers are scattered in two more or less distinct groups in the upper part of the pons. If it can be postulated that the portal of entry of the virus is via the afferents of the pharyngeal plexus, all of the above named structures are likely to be affected. Extension to adjacent structures, cervical cord below and upper cranial nuclei, hypothalamus, respiratory and circulatory centers above, would seem likely. The clinical course of the disease appears to follow such a process.[4]

The physiopathology of bulbar poliomyelitis demonstrates four derangements of function:

1. Dysphagia.

2. Involvement of the circulatory or respiratory centers.

3. Paresis or paralysis of the respiratory muscles.

4. Encephalitis or spinal involvement.

The usual course of the disease with its prodromal stage of fever, anorexia and malaise, followed by a temporary respite, recurring in three or four days with the ominous onset of meningismus is well known to all. Nasal voice and nasal regurgitation may then appear, followed by dysphagia. Thoracic and high cervical cord involvement may follow, manifested by weakness of the intercostals, diaphragms and limbs. Occasionally, however, the course may be one of spinal onset with a more or less rapid ascent to involve the bulb. These latter cases are usually much sicker and the prognosis worse than in those beginning as purely bulbar cases.

One of the most important clinical aspects of bulbar poliomyelitis is dysphagia. The swallowing mechanism is a complicated process requiring coordination of several central nuclei and the various muscle groups they control.[5] Edema, toxicity or disease may interfere not only with these nuclei but also with the association pathways between them.

Sjöberg,[6] of Stockholm, Sweden, emphasized two important clinical signs. First, patients with unilateral pharyngeal paralysis swallow best when lying on the healthy side. This he calls the *signe de rideau.* Barium swallow in vagotomized cats shows flaccid, wide pyriform sinuses filled with contrast medium. There is also noted a strong sympatheticotonic contracture of the cricopharyngeus muscle, an atonic dilated esophagus and spasm of the cardiac pinchcock. Esophograms of patients suffering with pharyngeal involvement demonstrate these findings. Bilateral laryngeal abductor paralysis often follows. Thus, the *signe de rideau* may forewarn trouble. Second, with paralysis of the anterior neck muscles, the sternothyroid, sternohyoid, omohyoid and scaleni, innervated by cervical segments one and two, it is clinically evident that the angle between the neck and the chin is much more acute. This is due to the contracture or tonus excess of the antagonists, the mylohyoid, digastric and stylohyoid innervated by the Vth and VIIth cranial nerves with the result that the hyoid bone and larynx are pulled upwards and backwards to the posterior pharyngeal wall blocking the hypopharyngeal airway. This *"angle sign"* is a clinically important warning that anoxia may be imminent. Involvement of the XIth nerve with paralysis of the sternocleidomastoid and trapezius muscles causes the neck to drop backwards and the shoulders forwards with the result that the cervical vertebrae are pushed forward, further occluding the airway. The danger of placing a tight respirator cuff around the neck of such a patient is obvious.

Normally there is about 1,000 to 1,500 cc. of saliva secreted daily, which may be increased to 2,000 by nervous disease, nausea or irritation.[7] Paralysis of the muscles of the tongue, palate and pharynx makes the second stage of deglutition impossible. This relatively tremendous amount of fluid must then pool at the most dependent portion of the patient's upper airway, depending upon his position. The battle the patient has between his desire for air and his effort to rid his airway of secretion not only tires and frightens him but also irritates his throat, increases the amount of secretion, may nauseate him or cause him to vomit. Vomiting may also be produced by interference with normal esophageal peristalsis due to direct involvement or irritation of the central nuclei

This secretion may act as a foreign body to the larynx and cause a partial or recurrent spasm of the cords.[8] The larynx may not be able to cope with this situation, and aspiration of the pharyngeal secretions is the result. Rarely there may be anesthesia of the larynx due to involvement of the superior laryngeal nerves. If this is the case, there is no "watchdog of the trachea," and the glottis is a functionally open door to pharyngeal secretions. A paralysis of one cord is uncommon and a bilateral abductor paralysis is relatively rare.

Dysphagia itself does little harm; however, secondary to it are respiratory obstruction and tracheal contamination with resultant anoxia and hypercapnia.[9] Secretional obstruction of the airway causes an increased intrathoracic pressure and has a *cupping effect on the capillaries whose walls are already* more permeable because of anoxia. This leads to congestion, stasis, edema, exudation, hemorrhage and even plugs with secondary atelectasis and infection Grave effects of the disease previously thought to be due to the direct action of the virus on the central nervous system, dyspnea, restlessness, antagonism, euphoria, mental depression, disorientation, drowziness, coma, increased blood pressure with later fall, vasomotor failure and circulatory collapse, are now known to be due to secretional obstruction and anoxia. Of all tissue, brain tissue is least able to withstand anoxia. Sixty seconds of anoxia produces distinct morphologic changes in the brain. Three to eight minutes produces widespread damage and necrosis of brain cells or death. Repeated periods of hypoxia may have an accumulative effect. Brain cells previously damaged by anoxia or infection are more susceptible to further damage by anoxia. Edema, hemorrhage or cell necrosis due to virus infection may block oxygen absorption. Profound and lasting effects of even brief periods of anoxia may lead to permanent mental and emotional change, imbecility, insanity, athetoid, Parkinsonian and even decerebrate states

Because of the danger of even a small degree of anoxia and hypercapnia to a brain and cord already damaged by a viral infection, efforts have been made to correlate the level of the blood oxygen and carbon dioxide to the patient's symptoms and clinical course. Oxygen levels from blood samples are too difficult to determine either accurately or repeatedly. Carbon dioxide combining power is a more applicable clinical test. A photoelectric oximeter attached to the ear lobe of a patient is reasonably accurate and determinations may be repeated at will. According to Gray and Seivers,[10] the narcotic properties of carbon dioxide are directly measureable. Concentrations of less than 10 per cent carbon dioxide of inspired air are not accompanied by symptoms. Those above 30 per cent will produce narcosis in most animals and above 40 per cent, pulmonary edema and hemorrhages from exposed mucous membrane surfaces. During the last war the air force demonstrated that a simple anoxemia of less than 60 per cent arterial oxygen saturation produced unconsciousness without symptoms. The restlessness, apprehension, distress and disorientation seen in asphyxia was thought probably to be due more to hypercapnia than to simple lack of oxygen. Gray also points out that acidosis may be a complication of asphyxia because of the retention of carbon dioxide and the excessive production of lactic acid in the muscles during anoxemia. Two interesting facts were found from these studies: first, that clinical signs of oxygen deficiency are evident only when anoxemia is far advanced; second, even with a normal blood oxygen level, a high blood carbon dioxide level has deleterious effects. Maintenance of a normal oxygen saturation of the blood by artificial means is, therefore, not enough. The airway has to be open to rid the blood stream of excess carbon dioxide.

Weakness or paralysis of one or both diaphragms due to high cervical cord involvement and ineffectual chest expansion due to thoracic involvement is so often associated with disturbance of the pharynx and larynx that they must be considered as part of the clinical picture of bulbar poliomyelitis even though they are not due to interference with the bulb itself. Weak, ineffectual cough, retention of aspirated material, and atelectasis complicated by infection may result.

For the sake of completeness, respiratory and circulatory center involvement is included. Baker[11] states that of 183 bulbar poliomyelitis patients, 36 were classified as belonging to the respiratory center group. Of these, 69.1 per cent died. Twelve were classified in the circulatory center group, and 83 per cent of these succumbed. Involvement of the respiratory center is indicated by irregularities of the rhythm and depth of respiration even though the intercostal muscles and diaphragms are functioning normally. Apprehension, confusion and delirium leading to coma gradually develop, accompanied by periods of apnea and Cheyne-Stokes respiration. Cyanosis becomes increasingly intense. In the bulbar circulatory group, patients have a dusky, red, flushed, florid appearance; the lips are a deep cherry red; the pulse becomes rapid, and the blood pressure has a downward trend; the patients are restless, anxious, apprehensive and at times confused, and fever is high.

Encephalitic symptoms, hyperexcitability, restlessness, irritability, mental confusion, irrationality, personality disturbance, insomnia or somnolence are frequent; however, most of these cerebral symptoms disappear on adequate oxygen administration and a clear airway. It would seem likely, therefore, that most cases previously classified as encephalitic in reality may be only anoxic.

Treatment: The therapeutic objective is a free and normal gaseous interchange in the pulmonary alveoli. To recapitulate: hypopharyngeal pooling of secretions due to involvement of IXth and Xth cranial nerves accompanied by weakness or paralysis of the intercostals and diaphragms is the chief factor with which we have to deal. Associated with and secondary to this are anoxia, dehydration, infection and fear.

The reasons for the accumulation, pooling and tracheal contamination of secretions have been discussed, and the complications resulting from these have been listed. The patient who can still swallow should be placed in the position easiest for him, while those who cannot should be placed in Trendelenberg position sufficient to clear the hypopharynx of secretions. Prone position with the head to one side takes care of the situation fairly well when this position is practicable. In the supine Trendelenberg position secretion will pool in the nasopharynx where it may be removed by intranasal suction. Aspiration should be done gently as it is irritating and may cause an increased amount of secretion or nausea. A word of warning should be given. Water suction has been known to back up and drown a patient.

Fluid intake should be somewhat low to keep secretions at a minimum but not low enough to cause dehydration. Atropine should not be used for obvious reasons. Food and fluids cannot be given by mouth. Two methods, parenteral and tube feeding, are then available. During the very acute stage of the disease, which as a rule lasts only a few days, intravenous fluids are the method of choice. Saline and glucose suffice in most cases; however, intravenous aminoacids or even plasma or blood may be indicated occasionally. Tube feeding has been frowned upon because of irritation which may cause increased secretion and vomiting. The tube should be passed into the stomach to lessen the possibility of regurgitation because of the atony of the esophagus and the spasm of the cardia. The paralysis of the swallowing mechanism, the flaccidity of the pharyngeal musculature, the spasm of the cricopharyngeus and cardia, and the atony of the esophagus may make passing the tube very difficult indeed. It may, however, be passed with the aid of a laryngoscope and forceps, an esophagoscope or esophageal speculum, on a stylette, or with the eye of the tube fitted into a fine flexible-tipped esophageal bougie. The new polythene plastic Levine tubes are less irritating then the rubber tubes. I have found, however, that polythene tubing, the lumen of which is large enough to admit only a No. 20 gauge needle, was tolerated very nicely by both child and adult patients without any irritation, discomfort, nausea or vomiting, and that a continuous slow drip could be attached to this tubing which would give the patient an adequate feeding. It is of much assistance in maintaining adequate nutrition and hydration without irritation, discomfort or nausea. This tubing is illustrated in use in the accompanying photographs (see Figs. 1 and 2).

If secretions are aspirated into the tracheobronchial tree before or despite the preceding measures, the lower airway will become obstructed by secretion and atelectasis, and infection will take place. The trachea of a patient lying supine is directly posteriorly at an angle of 20 degrees; therefore, at least 30 degrees of Trendelenberg position is necessary for adequate postural drainage. This amount is extremely difficult as the patient must be literally suspended by his ankles to prevent sliding up the bed. It is nearly impossible in a respirator. The cilia of the mucosa of the tracheobronchial tree will function much better if aided by gravity. Coughing should be encouraged. Intranasal tracheal aspiration is the easiest and least traumatic method of ridding the lower airway of

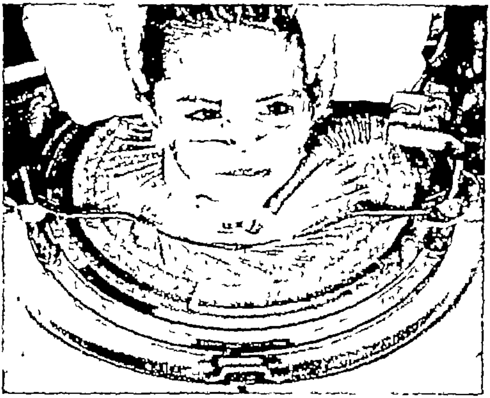

Fig 1 Three-year-old tracheotomized child in tank type respirator. Small plastic feeding tube, Monchan positive pressure apparatus, and Spira-twist plastic neck cuff illustrated in use.

secretions. It may be repeated often with little danger or discomfort to the patient. If this is not possible, direct laryngoscopy to pass the catheter may be used. Direct laryngoscopy also allows one to look for paralysis of the cords, test for laryngeal anesthesia, and observe how much of the respiratory obstruction is hypopharyngeal. If this seems impractical, due to a great deal of secretion, aspirated vomitus, or atelectasis, one may resort to bronchoscopy. I should like to give a word of warning: an anesthetized larynx is an open door to the tracheobronchial tree; therefore, bronchoscopy

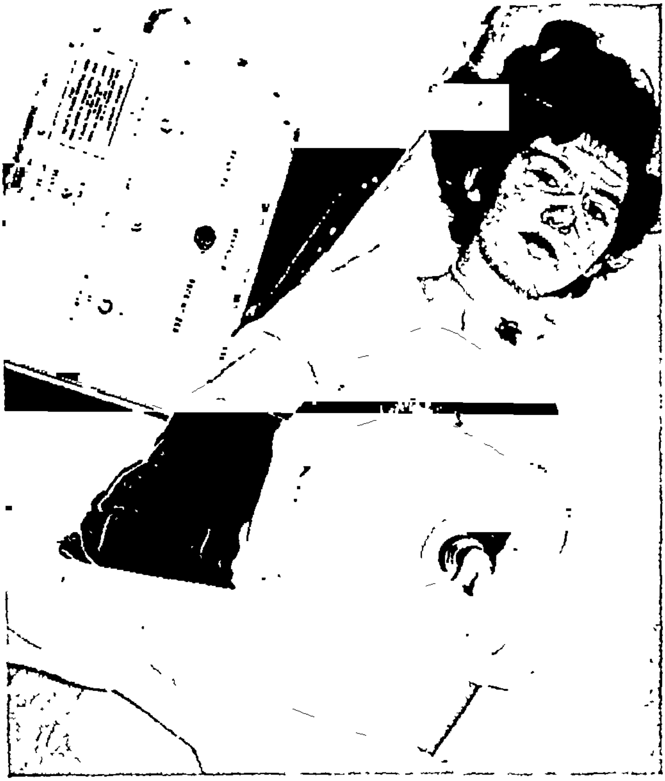

Fig. 2. Composite posed picture of a convalescent tracheotomized 14-year-old girl illustrating use of small plastic feeding tube, plastic bubble chest type respirator, and Respir-aid rocking bed.

or laryngoscopy should be done without local anesthesia if possible. Another warning: if bronchoscopy is necessary, I have found that laryngeal irritation, stimulation of a great deal of secretion (witness many bronchoscopies in normal patients), and rapid reaccumulation of pulmonary secretions may necessitate early tracheotomy.

Since there is a high incidence of atelectasis, patients should be encouraged to cough at frequent intervals, do deep breathing exercises, exhale through blow bottles or inflate rubber balloons. A face mask is available for the Monahan positive pressure apparatus, of which more will be said later. Through it, alone, or in conjunction with the respirator, room air or oxygen may be delivered at pressures regulated up to 20 mm. of mercury. This device will greatly discourage atelectasis if it seems imminent and will help to re-expand an atelectatic lung and reduce pulmonary edema.

There is an ever present danger of infection due to contamination of the tracheobronchial tree by aspirated pharyngeal secretions. These patients should be placed on prophylactic chemotherapy. Sputum cultures should be obtained as mixed infection is common. Unfortunately an infection with B. pyocyaneus is not rare. This organism is resistant to most therapy but will usually succumb to full and continued doses of streptomycin. Prophylaxis should be continued as long as there is any possibility of primary or recurrent pulmonary infection.

Restlessness, anxiety and fear are common and very real symptoms of bulbar poliomyelitis. Calm, quiet reassurance should take the place of sedatives when possible. Narcotics further depress the cough reflex and respiratory center and are contraindicated in the acute phase of the disease.

The most fundamental problem is getting an adequate amount of oxygen to the alveoli and removing the accumulated carbon dioxide. For this, a clear airway is essential. Supplementary oxygen may be given by face mask, intranasal tube or tent. The mask alone is not tolerated well over a long period of time. The catheter is better tolerated but may irritate, cause gagging and dry the secretions. The tent is most comfortable but is not as efficient in oxygen concentration. An oxygen-carbon-dioxide mixture may not stimulate respiration as a hypercapnia may already be present. The oxygen should be moistened by being passed through water, to which may be added sodium lauryl sulphate, U. S. P., a wetting agent.

Restlessness, anxiety, fear, air hunger, diminished expansion of the thoracic cage and diaphragmatic movement, increasing pulse and respiratory rate, pallor, and finally cyanosis are indications for artificial respiratory assistance. The simplest and most immediately available method is manual artificial respiration. Hand pumping the bag of an anesthesia machine connected to a tight fitting face mask makes an ideal temporary respirator; however, its most ideal use is during tracheotomy. The patient may be removed from the respirator and the operation done with ease without respiratory embarrassment to the patient or the inconvenience of a tank type respirator to the surgeon.

The tank respirator is universally the most satisfactory and efficient, but has certain disadvantages. Nursing care and administration of intramuscular or intravenous medication is difficult. The psychological effect on the patient may be bad. Lying supine with little chance of change of position and a tight chafing neck cuff is uncomfortable. The major problem, however, comes with tracheotomy when the neck cuff must be kept below the tracheotomy tube. The usual sponge rubber neck cuff is difficult to hold in this position but may be held by a long, thin surgical retractor attached with heavy tape to the top midsection of the respirator.[12] The Spiratwist plastic neck cuff with its readily adjustable lumen has been found eminently satisfactory. A special curved metal rod can be slid in place, holding this cuff easily in position. Tracheotomy care in the respirator is greatly facilitated by this efficient and easily adjustable device. This is illustrated in Fig. 1.

The plastic bubble chest type respirator[13] is much more comfortable, better tolerated by the patient, easier for nursing care, much more convenient in tracheotomized patients but is not as efficient in the more completely paralyzed patients. In mild and convalescent cases, as a temporary expedient, and in transportation its value is great (see Fig. 2).

The McKesson respir-aid rocking bed,[14] an adaptation of the Sanders oscillating bed, used to improve circulation in peripheral vascular disease, works on the principle of passive motion of the diaphragm, caused by shifting the abdominal contents in its up and down motion. The rate may be adjusted from 12 to 26 per minute and the depth of respiration controlled by the angle to which the bed is deviated. The passive motion of the diaphragm may encourage whatever weak phrenic stimuli are available. Frequency of atelectasis is diminished. Muscular tone and circulation are improved. The incidence of deep phlebitis in the lower limbs with secondary pulmonary embolism, a not too uncommon occurrence in these patients, is lessened. Its practical use is about the same as the chest type respirator with the added advantages listed (see Fig. 2).

The electrophrenic respirator developed in Boston may have limited use. The motor point of the phrenic nerve may be found by digital exploration of the angle between the clavicle and the sternocleidomastoid muscle with an electrode mounted on especially equipped rubber finger cot.

When found, contact may be maintained by taping the electrode to the skin in conjunction with external pressure. A metal mesh strap type electrode is available which may be wrapped around a surgically exposed phrenic nerve. The depth and rate of respiratory may be controlled by adjusting the intensity and the time interval between the electric stimuli. This apparatus may have a real place in cases of respiratory center failure.

Tracheotomy: A complete understanding of the pathophysiology of bulbar poliomyelitis should make the indications for tracheotomy self-evident. They will, however, be listed. Galloway suggests the following indications:

1. Progressive anoxia with secretions in the upper airway.
2. Rapidly progressive bulbar symptoms.
3. Fluid accumulation despite frequent aspiration in a patient who requires a respirator.
4. Pronounced restlessness or stupor in a patient in a respirator even if the paralysis is apparently of the spinal type.
5. Unconsciousness or pronounced restlessness in the patient who does not respond to other treatment in a few moments.
6. Grave signs of vasomotor failure.
7. Bilateral spasm or paralysis of the vocal cords.
8. Untrained or inefficient attendants, inadequate equipment, or poor patient cooperation with doubt that the airway will be kept free from secretions.

The Minnesota Poliomyelitis Research Commission states that "Elective tracheotomy is indicated when recent onset of bulbar symptoms with evidence of progression of illness, progressive difficulty in swallowing or accumulation of secretions in the throat, and mental changes, especially anxiety, apprehension, hyperactivity, confusion or euphoria occur." They further state that "The appearance of laryngeal stridor, dyspnea despite adequate chest expansion, cyanosis and severe encephalitic symptoms indicate serious obstruction of the airway and require emergency tracheotomy." Mechanically forced inspiration through a pool of mucus in the pharynx will draw it into the chest.[15] For this reason the respirator was for many years said to be contraindicated in bulbar poliomyelitis. If, however, tracheotomy is done, the air exchange by-passes this fluid and there is little danger of aspiration.

Little need be said about the technique or complications of the operation. The usual midline incision may, however, be replaced by a transverse incision, making the operation easier if done in the respirator, causing less chafing of the neck cuff on the wound, and giving more comfort and less residual scar to the patient. Subcutaneous or mediastinal emphysema and pneumothorax have not been reported as complications either with or without the respirator.

As usual in tracheotomy the after-care is much more important than the surgery. Constant, well directed nursing care of high quality is essential. The secretions in the mouth and nasopharynx should still be removed. The lumen of the tube should not be encroached upon by allowing secretion to dry on it. The tracheobronchial tree should be kept free of secretions by frequent suction. Cough should be encouraged. Since tracheotomy by-passes the moistening effect of the nasal mucosa, the humidity of the room should be kept high to avoid drying of secretions and crusting. The Walton cold steam humidifier is excellent. A few drops of saline or weak sodium bicarbonate solution dropped into the trachea discourages or may loosen crusts. Apprehension and increased pulse may be a warning that there is an obstructing mucus plug or crust which may require bronchoscopic removal. Prophylactic chemotherapy should be given. Oxygen administered directly into the trachea is drying. Moistening and administration through a funnel held over the tracheotomy tube will help to prevent this.

Pulmonary edema, atelectasis or air hunger despite the respirator may be benefited by positive pressure respiration. The Bennett positive pressure apparatus is excellent; it may be used alone or in conjunction with the respirator. Alone, it works on the same principle as hand pumping the bag of an anesthesia machine with a closed system. With the respirator it accomplishes respiration by the regulated and synchronized dual action of negative pressure within the respirator and its own positive pressure which delivers filtered humidified air or oxygen into the tracheotomy tube or face mask. The apparatus is attached to the bellows of the respirator to synchronize their independent action. Simultaneous positive and negative pressure respiration has reduced or eliminated pulmonary acidosis, dried up excessive tracheal secretions or pulmonary edema, and synchronized the patient's respiratory rhythm when disturbed by excess carbon dioxide accumulation or by a direct action of the virus on the respiratory center (see Fig. 1).

A simpler type of positive pressure may be devised by connecting an oxygen stream directly to the tracheotomy tube.[16] Not only does the pressure in the line tend to expand the lungs on inspiration, but since expiration must be accomplished against this pressure and around the tracheotomy tube through the larynx, the same effect as a blow bottle is achieved. Secretions in the hypopharynx at the same time are forced upward where they may be more readily removed. A similar system employs a special type of tracheotomy tube which has two lateral and one superior opening.[17,18] The superior opening is plugged except when suction is necessary. Positive pressure inspiration comes from a humidified, filtered oxygen source into one lateral opening; positive pressure expiration through the opposite lateral opening to a blow bottle. Either of the last two methods of positive pressure can easily be assembled in any hospital and are very adequate, although more difficult to maintain than the mechanical Bennett positive pressure apparatus.

The problem of when to decanulate a patient may be perplexing and trial and error may be the only solution. In general, however, it is best to wait until the patient is out of the respirator, has no more tracheal secretion, and can breathe comfortably for 48 hours with a regular or fenestrated tracheotomy tube plugged off. It is better to leave the tube in too long than to have to reinsert it through a tracheal stoma that has completely closed its lumen. Needless to say the psychic trauma of the latter is far worse than the former.

CASE REPORTS.

Case 1: G. B., a 34-year-old factory inspector, complained of back pain, fever and stiff neck for three days prior to admission. Upon admission his temperature was 100.4°. There was a fine tremor of the hands, marked nuchal rigidity and back stiffness. Spinal fluid showed 250 cells and 170 mg. per cent of protein. This progressed to quadraplegia with concomitant facial and pharyngeal paralysis. Four days after admission, he became disoriented and started to pool secretions in his pharynx He was bronchoscoped and secretions were aspirated, but they rapidly reaccumulated. He was placed in a respirator, but his respirations were asynchronous with the machine He was removed and a tracheotomy performed with marked improvement of cyanosis and mental changes His course was later complicated by thrombophlebitis and pulmonary embolism. He was treated with anticoagulants The patient was decanulated on the twenty-eighth day and is now doing well, although he exhibits extensive residual paralysis.

Case 2: M. R., a 36 year old Methodist minister, developed headaches, stiff neck, nausea and vomiting for five days prior to admission. Spinal tap at another hospital revealed pleocytosis. When examined he was found to be severely ill with a temperature of 102° His pharynx was filled with purulent secretion, and there was a right facial weakness and flaccid arms and legs He required constant suctioning, and within two days he was completely irrational Because of severe persistent cyanosis, irrationality and confusion a tracheotomy was performed The trachea was found to be free from secretions at the time of surgery It was felt that much of the hypoxia was secondary to prolapse of the tongue occluding the hypopharynx. The cyanosis and mental confusion cleared completely. He did fairly well except that his trachea had to be suctioned almost constantly. In spite of this, his temperature rose to 104°, and he had frequent episodes of cyanosis and air hunger. Positive pressure oxygen was given through the tracheotomy tube with considerable relief of these symptoms. His tracheotomy tube was removed on two occasions but had to be replaced because of recurring secretional obstruction He was permanently decanulated on the sixty fifth day. He was gradually removed from the respirator to the rocking bed and then to a regular bed without respiratory difficulty Both arms and both legs were completely paralyzed. He had residual facial paralysis and some weakness of his pharyngeal musculature On the one hundred and twenty-first day he became weak, confused, had a little mucus in his throat and died suddenly three days later Postmortem examination showed his tracheobronchial tree to be clear of secretions and only a tiny patch of atelectasis in the base of the left lung There was very extensive destruction of the anterior horn cells with actual canulization of the cord.

Case 3: D C, a seven-year-old white female, had been ill five days with nausea, vomiting, stiff back and nasal regurgitation. Temperature rose to 103°. She spoke with a nasal twang There was left facial weakness and marked nuchal rigidity, 164 cells and 53 mg. per cent protein in the cerebrospinal fluid. Her restlessness progressively increased. Her respiratory rhythm was not smooth There was a moderate amount of secretion in the pharynx. Tracheotomy at this time was suggested. It was refused by the attending physician, but six hours later tracheotomy was done because of sudden cyanosis due to bilateral abductor paralysis The patient went into peripheral shock with Biot breathing and died four hours later.

Case 4: L. B., a three-year-old male, was found to be ill five days before admission when he awoke crying Three days before admission he was unable to move his arms or legs. Physical examination revealed a temperature of 100°. There was slight flaring of the alae nasae and there was moderate respiratory distress with little chest motion. All four extremities were weak. Spinal tap revealed 130 cells and 48 mg per cent of protein. On the second hospital day, his respiration became irregular and feeble. He was placed in a respirator and given intranasal oxygen.

The next day he had two convulsions and then became comatose. Direct laryngoscopy revealed a normal larynx without paralysis or edema and very little secretion. Because of a progressive downhill course, a tracheotomy was done. Very little mucus was found in the trachea; however, the patient's color immediately improved, and further course was uneventful. He was decanulated on the fifty-seventh day, and at the present time he uses a rocking bed about half of the time, the rest of the time being in the tank type respirator.

Case 5: M. R., a 14-year-old female, had been vomiting for four days and developed a nasal twang to her voice with marked nasal regurgitation. There were 94 cells and 39 mg. per cent protein in the spinal fluid. Positive physical findings were a temperatuer of 104.5°, blood pressure of 140/90, and much pooling of mucus. She was able to speak only in a whisper. There was only minimal intercostal motion, but she was well oriented. She was suctioned, placed in an oxygen tent and seemed to improve. Because pooling continued and respiration became more irregular with periods of apnea, a tracheotomy was performed. She was placed in a respirator for only 12 hours. Her subsequent course was good with the aid of a rocking bed. The tracheotomy tube was removed 10 days later. She was discharged from the hospital with only a slight nasal voice and no other residual paralysis.

Case 6: J. N., 16-year-old white male, complained of headache, stiff neck, and a weak left leg. Spinal tap at another hospital showed 104 cells with a positive Pandy. He developed weakness of the right arm and leg and some respiratory distress. He was, therefore, transferred to Syracuse. His temperature was 103°. He was apprehensive and had a very weak cough with many coarse rales in the right lung. Two days later he was found to have complete atelectasis of the right middle and lower lobes. He was bronchoscoped, and a fluid level was found just below the right upper lobe orifice. After this secretion was removed sounds could be heard coming through the lower portion of the right lung. Soon, however, there was progressive respiratory difficulty with reaccumulation of secretions. Tracheotomy was done, and a great deal of mucus was found in the tracheobronchial tree. He immediately improved after tracheotomy. A moderate amount of secretion continued and was aspirated. Gradually the amount of these secretions reduced. He was decanulated on the thirteenth day. His subsequent course was uneventful.

Comment: It seemed quite definite that tracheotomy was of great value in these cases. Of these six patients, two died. The first patient died of late complications long after he had been decanulated. It was felt that the second might not have died had an earlier tracheotomy been done. It is my feeling that the other patients would certainly have died had not tracheotomy been done. Of the 68 cases of bulbar poliomyelitis only six died. Two have been mentioned. Three died suddenly without obstruction of the airway, apparently due to involvement of the circulatory center. Another died of a very rapidly progressive bulbar type of disease. Of a total of 20 cases demonstrating partial obstruction of the airway, tracheotomy was done in only six because of the other measures mentioned and a careful selection of cases.

Summary: The pathophysiology, clinical course, signs, symptoms and complications of bulbar poliomyelitis have been discussed in detail. An attempt has been made to correlate treatment with altered physiology. The therapeutic armamentarium that is available has been described. Indications for tracheotomy have been reviewed, and six cases requiring tracheotomy in the 1950 epidemic in Syracuse, N. Y., have been presented.

Conclusions: The most important symptom of bulbar poliomyelitis is the inability to swallow. Accumulated secretions obstructing the airway and contaminating the tracheal bronchial tree, anoxia and hypercapnia are the result. Tracheotomy, if done before anoxia has produced serious central nervous system damage employed in conjunction with other therapeutic measures, gives the patient the best chance of survival until whatever natural recovery of damaged neural tissue can occur.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

June 1, 1951.

Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.

Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico.

Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Model R-2.

Manufacturer: The Microtone Co., Ford Parkway on the Mississippi, St. Paul 1, Minn.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer Micronic Corp, 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.

Manufacturer National Hearing Aid Laboratories, 815 S Hill St., Los Angeles 14 Calif

Distributor Sears-Roebuck & Co., 925 S Homan Ave., Chicago 7, Ill

Silvertone Model J-92

Manufacturer Sears-Roebuck Co, 925 S Homan Ave, Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer Micronic Co, 727 Atlantic Ave., Boston 11, Mass
Distributor Sears Roebuck Co., 925 S Homan Ave, Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer W E. Johnston Mfg. Co., 708W 40th St., Minneapolis, Minn.

Distributor Sears-Roebuck & Co, 925 S Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer Solo-Pak Electronics Corp, Linden St., Reading, Mass

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer Sonotone Corp., Elmsford, N Y

Superfonic Hearing Aid.

Manufacturer American Sound Products, Inc., 2454 S Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer *Televox Mfg Co, 117 S Broad St., Philadelphia 7, Pa.*

Telex Model 22, Telex Model 97; Telex Model 99; Telex Model 200, Telex Model 300B; Telex Model 400, Telex Model 1700.

Manufacturer Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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SOME ASPECTS OF INNER EAR THERAPY.*

JEROME A. HILGER, M.D., and
NEILL F. GOLTZ, M.D. (by invitation),
St. Paul, Minn.

From the therapeutic standpoint the disorders of the inner ear have constituted for otologists a realm of defeatism and impotence. The past two decades have seen important contributions in the qualitative and quantitative analysis of inner ear disorder and have seen salient electroacoustic advances in the supplanting of accepted hearing impairment by amplifying aids. Prevention of hearing impairment has received accent, but this has been largely limited to the preservation of the middle ear in school age and the reduction of acoustic trauma in industry. The idiopathic realm of inner ear impairment while it comprises the bulk of otic disability has been largely barren of therapeutic advance. This is reasonable and right. A realm in which pathology is known but pathogenesis is unknown can hardly be graced by therapeutic advance.

Otology has long confused itself and dissipated its energy in a welter of unbasic theses of primary neuritic, heavy metallic, outdated luetic, and meaningless catarrhal hypotheses which led to the dead end of therapeutic nihilism.

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*Read at the Fifty-fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 7, 1951.

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There is every reason to believe that an important segment of inner ear pathology translated during development into symptoms of tinnitus, hearing alteration, and vertigo results from vascular change in the end-arterial distribution of the labyrinthine artery. While some of this vascular change is predicated upon histologically demonstrable atherosclerosis, endarteritis and similar obliterative change, the usual alteration is not in vascular character but in vascular function. It represents autonomic dysfunction in the labyrinthine neurovascular sphere. The penalty for angiospastic change in the labyrinthine arterial tree is severe because the primary neurones of the inner ear are most intolerant of resulting ischemia and because the vascular anatomy of the labyrinth is ill-designed for the resorption of the transudate resulting from ischemic damage to the capillary.

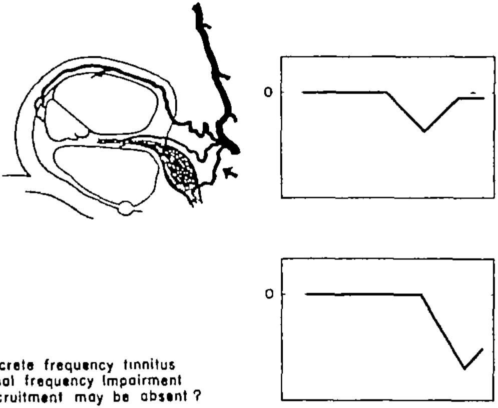
SYNDROMES.

The clinical syndromes resulting from labyrinthine angiospastic change vary with the neural segment involved and the degree of involvement. They may be the picture of chronic progression due to minute, insidious and continuing neurovascular dysfunction; they may be the sudden, dramatic, all-encompassing disaster involving acoustic and static function in colossal ischemic disruption. The syndromes must be viewed and taught as variations of a single genesis — differences being due only to degree of ischemia and nature of labyrinthine elements involved. The resultant theme brings order to this vast segment of clinical otology. It points the way for therapy.

The syndromes have been elaborated before. It is well to reiterate them now before relating them to specific case histories and manner of therapy. The syndromes themselves are a matter of clinical observation. The designs of their origin are hypothetical — a graphic attempt to present a mechanism of formation for known clinical phenomena.

Primary cochlear ganglion cell ischemia (see Fig. 1) causes specific frequency tinnitus and zonal hearing impairment.

There is no distortion with diplacusis, no aural pressure, and no vertigo. It eventuates in the so-called "VIIIth nerve neuritis" or "catarrhal deafness" of yesterday.



Discrete frequency tinnitus
Zonal frequency impairment
Recruitment may be absent ?

Fig 1 Ischemia of the cochlear ganglion cells (catarrhal deafness VIIIth nerve neuritis, etc.)

Cochlear stria vascular ischemia (see Fig. 2) results in endo- and/or perilymphatic hypertension because transudate through the damaged capillary endothelium finds itself in the labyrinthine space since the capillaries of the stria are unique in that they are covered only by the epithelium of the cochlear duct or the mesothelium of the scalae Tonal distortion and aural pressure are experienced before hearing impairment is measurable. The presence of aural pressure and the rising curve of the audiometric tracing readily imply middle ear or tubal pathology. Thorough otologic examination and bone conduction testing are essential for differentiation. Onset vertigo is common. Tinnitus may be absent or may be of a broad frequency varying type. Without vertigo this again is the "catarrhal deafness" of old with a middle ear implication or "the endolymphatic hydrops without vertigo" of more recent vintage. With vertigo this symptom complex ofttimes came under the coverall of "Ménière's disease" or "syndrome."

The vertigo of stria vascular ischemia results from the distortion of the vestibular neuroepithelium at the onset of lymphatic hypertension. If the latter develops exceedingly gradually or is confined to the ductus cochlearis by anatomic block of the utriculosaccular valve or other device vertigo may not be experienced at onset. It is not uncommon, however, to encounter instances where lymphatic hypertension confined to the cochlear ducts for months with resultant cochlear disability suddenly diffuses through such anatomic block to the vestibular spaces with a resultant sudden vertigo. In some instances this sudden diffusion and dilution of cochlear lym-

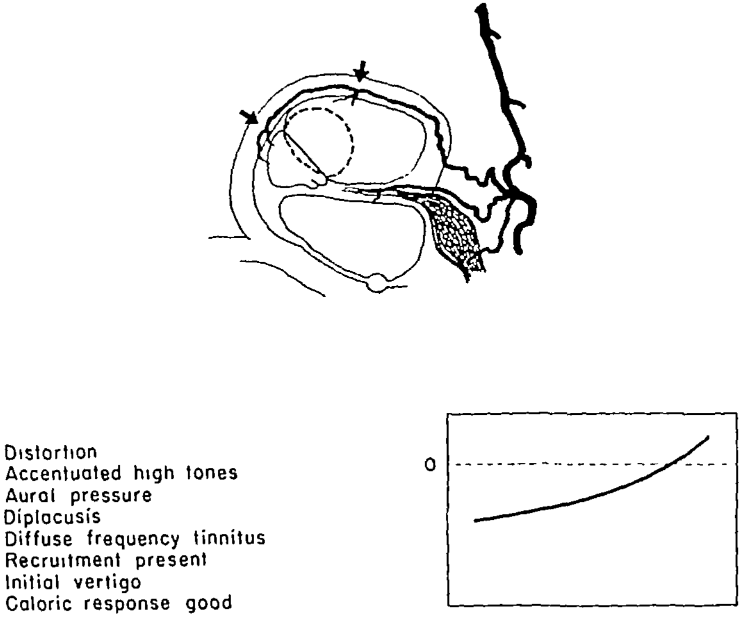
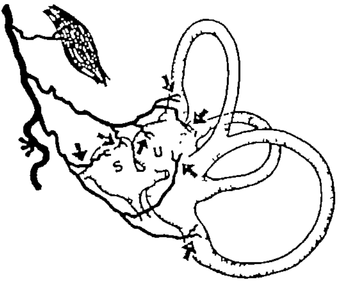


Fig. 2. Ischemia in the stria vascularis with formation of endo- and/or perilymphatic hypertension (Ménière's disease, endolymphatic hydrops with vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops without vertigo).

phatic hypertension into a large space results in a decrease of tension and distortion in the cochlear duct and scalae with resultant gain in hearing following the severe vertigo (Cases IV and X).

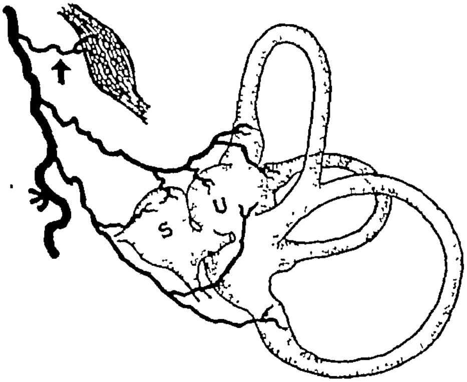
Macular or ampullar ischemia (see Fig. 3) produces a vertigo of specific type dependent upon the neuroepithelial plaque involved. It can occur without cochlear involvement and fall under the former blanket of "pseudo-Ménière's disease." It is interesting and noteworthy of the punctuate character of angiospastic change in the labyrinth that, following the onset of a discrete macular or ampullary ischemia and damage the patient's neuroepithelial disability is translated for weeks or months as "dizziness" initiated by movement in the specific plane of that end-plate.



Vertigo (persistent with movement in the affected plane)
Caloric response impaired (if end-plate damage is
severe and test is discriminative)

Fig 3. Ischemia in macula or ampullary crest (pseudo-Ménière's disease).

The vestibular ganglion cells distributed along the course of the vestibular nerve have a richer interlacing of arteriolar terminals than is found elsewhere in the labyrinth. Less vestibular ganglion cell angiospastic ischemia may result because of this collateral support. When ischemia occurs in these primary neurones (see Fig. 4), however, the vertigo needs must be unconfined to end-plate distinction but seen in any and many planes. Until more widespread labyrinthine involvement occurs to allocate this vascular dysfunction to the end-organ there is no possibility clinically of differentiating this lesion from similar change in the central nuclear areas. It again has fallen under the looseness of "pseudo-Ménière's disease."



Vertigo (not confined to a discrete plane of movement. Often persistent without movement)
Caloric response impaired (if neural damage is sufficient)

Fig. 4. Ischemia in vestibular ganglion cells (pseudo-Ménière's disease).

As in segmental angiospasm anywhere in the body, the degree and spread of neurovascular dysfunction is individual. The pure form of ischemia confined to a discrete vascular segment in the inner ear is the exception — not the rule. Clinical cases frequently tend to be predominantly cochlear ganglionic; or predominantly stria vascular; or macular or ampulla; or vestibular ganglionic, but the relative variations are unlimited. The eventual trend is for more widespread neurovascular dysfunction and the total syndromes are common (see Figs. 5 and 6).

THERAPY.

There are three therapeutic approaches which must be balanced in any case: *A*. The instigating factors of neurovascular dysfunction must be obviated. *B*. Neurovascular tone must be brought to normal balance. *C*. The agents of repair for ischemically damaged tissues must be made available.

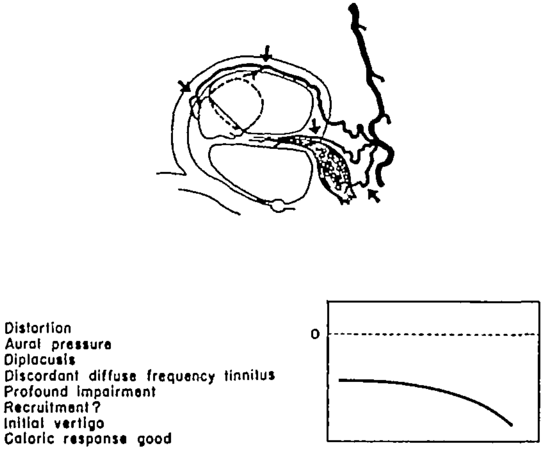


Fig. 5. Total cochlear ischemia (Ménière's disease, endolymphatics hydrops with vertigo, labyrinthosis, etc.; or, if endolymphatic hypertension is not transmitted through utricular valve—endolymphatic hydrops without vertigo).

A. The instigating factors of neurovascular dysfunction are those environmental factors to which the individual has come to react in an unfortunate, aberrant manner. Some individuals are more prone to this unfortunate reaction than others because of their inherently unstable autonomic nervous mechanisms. For this reason the family history and prior history of the victim of inner ear ischemia affords tell-tale background of other autonomic dysfunction which in the head segments is prone to fall into the realms of vascular head and neck pain, cervical and pharyngeal myalgia, vasomotor rhinitis, or vasomotor labyrinthine ischemia.

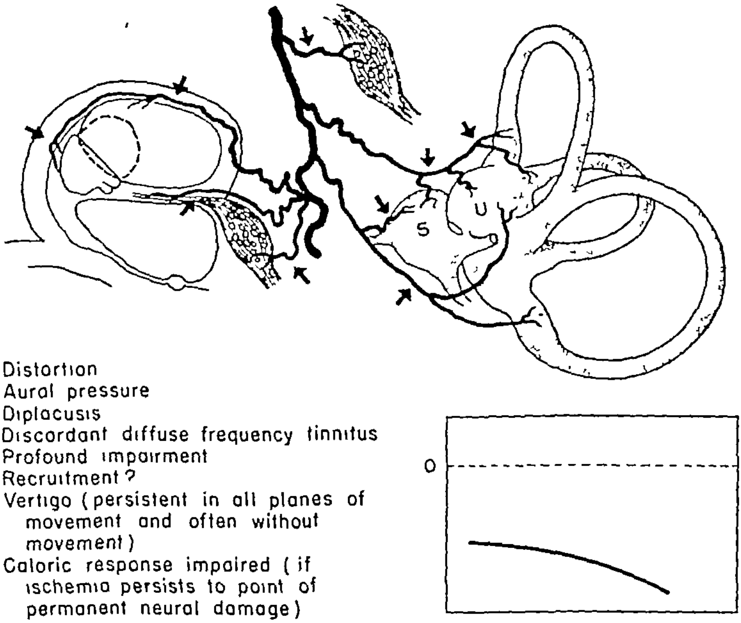

S
U
Distortion
Aural pressure
Diplacusis
Discordant diffuse frequency tinnitus
Profound impairment
Recruitment ?
Vertigo (persistent in all planes of movement and often without movement)
Caloric response impaired (if ischemia persists to point of permanent neural damage)
0

Fig. 6. Total labyrinthine ischemia (Ménière's disease, endolymphatic hydrops with vertigo, labyrinthosis, etc.).

The environmental factors which precipitate a specific neurovascular inner ear crisis are on only rare occasions autonomic-mediated antigen—antibody reactions from *inhalant or ingestant exposure* — so-called allergic crises in the inner ear. Infinitely more commonly the crisis is occasioned by unfortunate reaction to *physical environmental change.* The most common precipitor of vasomotor inner ear crisis, however, is upset in the fourth environmental sphere—*emotional reaction.*

The autonomic mechanisms are ordinarily prepared for the breakdown of crisis, moreover, by the vitiating factors of contingent stress — fatigue, coincident infection, and vasospastic agents and situations such as overindulgence in tobacco, coffee, or the pendulum-swing of excessive alcohol.

Taken individually or coincidentally these many factors impinging upon an inherently faulty autonomic mechanism conspire to the neurovascular crisis of the inner ear. *All of these factors must be weighed and neutralized as the first step in therapy.*

B. Beyond *the unmatchable tendency of nature to restore neurovascular balance* once the patient is put physiologically right with his environment there are *agents in the puny realm of drug therapy* that contribute to the same end. In general these pharmacal helps fall into two categories — *blocking agents* and *smooth muscle dilators.* The one neutralizes the neural impulse to spasm; the other actively counters spasm at the smooth muscle — principally at the arteriolar sphincter. The literature is full of haphazard and individual reference to these many agents, but cohesion and common ground is too little dwelt on.

The blocking agents — a large and rapidly growing field of interesting drugs:

1. At ganglion level there are principally tetraethylammonium chloride and direct ganglion infiltration (in our instance principally the superior cervical sympathetic ganglion with procaine, dolamine, etc.). Clinical use is limited except for the inquiring and academic mind.

2. At myoneural junction a whole host of drugs take blocking action: The so-called antihistaminics, artane, banthine, dibenamine, priscol, procaine, procaine amide, as well as atropine, hyoscine, ergotamine, etc. For the neurovascular inner ear crisis the degree of ischemia and the duration of ischemia determine the amount of permanent and residual neural damage; hence the crisis is a medical emergency. Aggressive blockade is reasonable. Our preferred agents are intravenous hyoscine as a stop-gap and intravenous procaine for prolonged infusion. To maintain the teetering neurovascular balance once a crisis is past we prefer artane and occasionally benadryl or dramamine orally.

Dosage schedules for intravenous blockade vary naturally with age, condition, and weight. Basic average doses afford some takeoff point:

1. Hyoscine hydrobromide 0.3 mg. well diluted and infused slowly intravenously is usually well tolerated.

2. Procaine hydrochloride in 0.2 per cent solution (dissolved just before use in 5 per cent glucose — or 10 per cent glucose if lymphatic hypertension is present) can be infused in total first dose of 4 mg./kg. calculated for the individual patient at a rate which is comfortably tolerated but which gives sign of useful block — slight thickening of tongue, slight feeling of float, etc. Usually this strength started at 30 drops per minute can be quickly altered in rate to accomplish this end with complete safety. The "block" should be maintained continuously for one and one-half hours to two hours once a day or twice a day as the case merits. The desired period of block naturally should be the dominant consideration, not total milligrammage in the infusions subsequent to the introductory first.

Oral dosage of artane or of benadryl or dramamine varies widely. The objective is useful block with minimum side effects. This generally entails six to eight-hour interval dose and sometimes necessitates the substitution of one drug for another because of disagreeable by-effects,

An initial 2 mg. of artane three to four times a day can be increased or decreased as need be. In instances where it is not tolerated or doesn't produce vasomotor symptom control dramamine or benadryl in recognized dose can be used alternatively.

After an angiospastic episode oral control may have to be continued for weeks or months to inhibit the usual tendency to recurrence.

It should be accented that in most cases vasomotor labyrinthine ischemia is unfortunately not seen in the acute angiospastic crisis. The otologist in his geographic orbit can educatively correct this in the coming years. Unless the patient is seen during or shortly following the crisis or subsequent aggravant recurrences there is no neural residuum from the ischemia to be regained by intravenous blocking measures. To apply such aggressive therapy to established neural residua would be gross overtreatment. The patient having residua from an angiospastic crisis in the recent past deserves, on the other hand, consideration from the standpoint of a prophylactic regime designed to prevent the usual trend to future recurrence. This embraces phase one of therapy — the correction of those environmental and contingent factors that instigate neurovascular dysfunction—and continuous or intermittent oral therapy that will tend to this same end.

Smooth muscle dilating agents are more familiar in average therapy than is appreciated: nicotinic acid, roniacol, the nitrites (sodium, amyl, nitroglycerin), ethyl alcohol, trichloroethylene, papaverine, histamine, adrenalin (in physiologic concentrations), etc. Our preferred agents for the acute crisis are intravenous papaverine or intravenous sodium nitrite for the stopgap and nicotinic acid added to the procaine solution for prolonged infusion. To maintain a vasodilating tendency over an extended period of oral maintenance therapy, nicotinic acid, or, where untoward side effects prevent its use, B pyridyl carbinol (roniacol) are preferred. Modest, therapeutic doses of alcohol are useful also.

Nicotinic acid is added to the intravenous procaine infusion in a trial initial quantity of 50 mg. to the 250 cc. In subsequent infusions this amount is varied up or down in the individual to correlate "mild float" with "mild flush" as a well balanced block and dilation.

Adrenalin may be added alternatively or additionally to nicotinic acid in the infusion in amount of 0.025 mg. to the 250 cc. of diluent as a physiologic vasodilator which also has the merit of increasing heart action.

Averagely nicotinic acid or roniacol are used orally four to six times a day. To stabilize dosage in relatively unstable reactors, they should be taken on an empty stomach, and the initial 50 mg. dose should be increased or decreased to a level giving occasional dilating sign.

C. *The agents of repair for ischemically damaged tissue* are both exogenous and endogenous. The basic facts of their mode of action are little understood. The first cells suffering ischemic damage are not the neurones of the end-organ but, rather, the endothelial cells lining the capillaries. The misfortune of transudate results. It can be said that early restoration of the capillary endothelium is the goal of repair therapy. The cells of the end-organ will inevitably restore within the limits of irreversible change if the integrity of the capillary is re-established.

Exogenous reparative agents are principally ascorbic acid, vitamin B factors, and vitamin E.; ✓vit A.

Asorbic acid is added routinely to the procaine infusion in amount of 1 mg. per cc. of diluent. There is evidence that it increases the tolerance to procaine and there is a mass of data available to demonstrate its effectiveness in endothelial repair.

Two hundred and fifty mg. to a gram of ascorbic acid is combined with the vitamin B complex for daily oral maintenance.

We as yet have little background for commenting on the use of vitamin E.

Endogenous reparative agents within the realm of our knowledge are principally thyroid and the steroid hormones.

Hypothyroidism of less than myzedema gravity is a clinical commonplace. It frequently is not a single entity but rather one evidence of a disordered endocrine chain. The whole picture of this complex is beyond our present horizon, but, at least, thyroid extract is a supplemental agent which is available to us, though it may not be the complete answer. When used in the rôle of a therapeutic accessory in the patient having general hypothyroid evidences it has a place. We have found the patient's symptoms of fatiguability, chilling, sluggishness, skin thickening, lip puffiness, etc., to be more helpful in its indication than the basal metabolic rate.

We cannot yet define the place of cortisone or the adrenocorticotropic hormone. It is our impression that it will have place in aborting recurrences in stria vascular ischemia in the early stages of development of lymphatic hypertension.

During certain age periods and under some circumstances of endocrine imbalance or absence, supplemental male or female sex hormones are essential to stabilize neurovascular function. This is particularly true at the climacteric and when ear symptoms of pressure, impairment, tinnitus, etc., are exacerbated regularly in relation to the menstrual cycle.

Five to 10 mg. of methyl testosterone daily in some recurrent labyrinthine ischemic patients in the male climacteric or 0.1 to 2 mg. of diethylstilbestrol in the female climacteric can produce vasomotor stabilization not otherwise obtainable. Where the vasomotor aural symptoms aggravate consistently in the last two weeks of the menstrual cycle, 10 mg. of methyl testosterone daily during this period for one or two consecutive cycles can be corrective.

Specific endocrine dosage varies so widely with the patient and the product that the individual otologist should provide himself with a background of his own reading or from his consultant endocrinologist before embarking on this phase of treatment.

CASE REPORTS.

Following are reports on cases illustrating the various types and combinations of types of vascular ischemia of the inner ear. Special otologic examination made repeatedly in each case during the course of the disorder ruled out the middle ear as the source of symptoms and impairment by means of frequent tubal recheck and tuning fork and/or bone conduction audiometric testing.

Where pertinent systemic physical conditions coexisted with the labyrinthine disability notation will be made. General examination including cardiovascular system, serology, fundi, cranial nerves, etc., may be assumed to present no related findings unless specifically mentioned. Pertinent reference to situations of physical or emotional stress seemingly so important in the production of or exacerbation of these conditions will be occasionally made.

Since in most of these cases during the acute phase of the vasomotor crisis repeated intravenous injections of 250 to 500 cc. of a 0.2 per cent solution of procaine hydrochloride in 5 or 10 per cent dextrose solution with one-twentieth mg. of adrenalin hydrochloride and 500 mg. of ascorbic acid were used, reference to such therapy for purposes of brevity in the case reports will be merely as "intravenous procaine." In some of the cases 50 mg. of nicamin replaced the adrenalin in the mixture, and where such was the case due notation will be made. Many other oral and intravenous vasodilators and blocking agents have also been used as substitutes for or in conjunction with intravenous procaine. We feel that each of the pharmacologic agents mentioned on the previous pages have due place in inner ear therapy. One cannot be used routinely to the exclusion of the others.

Vasomotor Ischemia of the Cochlear Ganglion Cells (see Fig. 1).

Case 1: A. G., female, age 23, complained of steady tinnitus in the left ear of several weeks' duration. Her middle ears were normal. The tinnitus was similar in tone to the high frequencies which were very minimally down on the audiogram (see Fig. 7, upper). She was given one intravenous procaine injection. The tinnitus disappeared and did not return. Fig. 7 (lower) shows the audiogram four days later.

Comment: The amount of involvement here is minimal and such cases are frequently neglected because of the lack of associated hearing loss or vertigo. As ischemia increases in severity and extent, permanent disability develops.

Case 2: This is a more severe example of cochlear ganglion cell ischemia.

A. R., male, age 45, had suffered from chronic vasomotor rhinitis for 20 years. On March 30, 1950, the patient complained of tinnitus of one day's duration in the left ear. The audiogram (see Fig. 8, upper) showed significant inner ear impairment on the left. He was given three intravenous procaine injections plus oral niacin during a period of 10 days. By April 10, 1950, the tinnitus was gone, but the audiogram (see Fig. 8, lower) was still somewhat down Oral niacin was continued He drove much of the subsequent night through a storm and the following night attended a party until midnight. The next morning after this fatiguing activity he had sharp tinnitus on awakening, and his audiogram was down again (see Fig 9, upper) Intravenous procaine promptly relieved the tinnitus except for minimal background noise. After two additional intravenous procaine injections he had to listen very carefully for the finest of tinnitus and his hearing was virtually normal (see Fig 9, lower) He was advised to continue oral niacin for a safety marginal six weeks and to use it intermittently thereafter at premonitory sign of vasospastic recurrence.

Vasomotor Ischemia in the Stria Vascularis (see Fig 2)

Case 7 A long standing repeatedly troublesome case for which labyrinthotomy or streptomycin destruction of the labyrinth was proposed Fortunately, however, changing to a less rigorous and tension producing job has alleviated her symptoms.

T. R., female, age 42, was first seen on April 8, 1948 She gave the history of onset of bilateral tinnitus and pressure sensation in the ears without vertigo in July, 1947 At that time she was seen at another office and this audiogram obtained (see Fig 10, upper) She was treated by inflation and nicotinic acid during the winter months of that year with improvement in the tinnitus on the right but persistence on the left. She was first seen by us seven months later (see Fig 10, lower) Examination revealed her drum heads and middle ears to be normal. After eight intravenous histamine injections plus oral niacin the tinnitus was less, although it was still quite constant on the left, and the hearing was improved (see Fig 11, upper) Subsequent to an extreme chilling, she developed a sharp increase in the tinnitus (see Fig 11, lower) Communication with her internist revealed he had previously given her thyroid, because of other hypothyroid symptoms. She was told to resume thyroid as well as to continue her oral niacin, and drop an overload of extra curricular activities. She was next seen seven months later, at which time she had only occasional tinnitus in the left ear when exposed to cold An amazing return of acuity was noted (see Fig. 12, upper).

Six months later on return examination she complained that she had for some time noted fluctuating but continuous tinnitus, diplacusis, aural pressure, and distortion of sound in her left ear Symptoms usually increased just before and during her menstrual period Mild vertigo and *some peculiar nausea and vomiting were recently present. She had had* some depreciation of hearing in the right ear as well as the left (see Fig 12, lower) She was given daily intravenous procaine and one-tenth mg. of diethylstilbestrol daily orally, together with ascorbic acid, vitamin B complex, and niacin. In less than a week her head symptoms had cleared completely, but several days later in bearing an overload of special responsibilities she developed a roar in her left ear, distortion, and pressure sensation again She again was given daily intravenous procaine injections in addition to her oral medications and 1 cc. mercuhydrin was added intramuscularly every fourth day in an effort to reduce the lymphatic hypertension more promptly After nine intravenous injections her ears had cleared (see Fig 13, upper) A month later her hearing dropped down again to the 30 db level in spite of the continued use of her oral medications. After five intravenous procaines the hearing was normal and diplacusis gone

After a vacation trip to Europe she returned to her strenuous work and in a matter of weeks she again developed tinnitus, this time bilateral, with aural pressure and hearing impairment in the left ear (see Fig. 13, lower). There was no vertigo. She also at this time developed asthma for the first time in her life. Oral niacin, ascorbic acid, diethylstilbestrol, and B complex hadn't prevented the return of ear symptoms. She was told to discontinue the vasoconstrictor histaclopane which she had been using for her asthma and was given 100 mg. of cortisone daily for four days. Four days later her asthma was vastly better. She was maintained on cortisone daily for three days and then on alternate days for an additional three weeks, but the lymphatic hypertension with tinnitus, hearing impairment, and aural pressure was unrelieved.

One month later she was hospitalized for two days because of an acute vasomotor upset — this time in the right labyrinth — with pressure sensation, tinnitus, and vertigo. She was readily controlled by procaine, but symptoms recurred in a week. She was again controlled with two intravenous procaine injections. She continued oral maintenance thereafter with artane, niacin, and diethylstilbestrol.

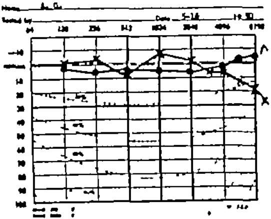
Because of these later two episodes of vertigo in the face of her continued use of oral medications she sought consultation elsewhere and streptomycin destruction of the labyrinth or labyrinthotomy was suggested. This was not followed through, however. Instead she continued oral medications, and prophylactically took, in addition, sodium nitrite 100 mg. intravenously at the suggestion of trouble. At long last she acceded to our repeated insistence that she get rid of her duties as a charge nurse. Six weeks after surgery had been suggested and refused it was apparent that her lighter program and oral medications were relieving the lymphatic tension (see Fig. 14, upper). She felt in excellent general health, and the tinnitus and aural pressure were lessening. She is maintaining on 3 gr. of thyroid daily, niacin, B complex and diethylstilbestrol. When on rare occasions she feels the slight nausea which usually precedes her vertigo she checks it with artane. Her audiogram is practically at normal now (see Fig. 14, lower).

Case 4: This case of vasomotor ischemia in the stria vascularis will be briefly discussed without detailed reference to day to day therapy, as it was in general quite similar to that used in the previous case. Note again the episodes of marked depression of hearing in the lower tones with return to normal even after prolonged periods of time.

E. T., female, age 60, was first seen in June of 1948, complaining of tinnitus, diplacusis, and hearing loss in the left ear (see Fig. 15, upper). This had been present for five days and had been associated with vertigo, nausea, and vomiting at the onset. She gave a history of a similar episode occurring two years previously which had cleared spontaneously over a period of many months. This time she was given intravenous histamine treatments and oral nicotinic acid and cleared of her symptoms during the next two weeks. She remained clear for another two years (see Fig. 15, lower) save for an occasional transient minor episode of vertigo sometimes associated with minor temporary tinnitus.

In May of 1950 she had an abrupt return of tinnitus and aural pressure in the left ear (see Fig. 16, upper). Daily intravenous histamine and procaine injections were used alternately. About two weeks after the onset of the tinnitus and auditory block the patient experienced a severe spell of vertigo lasting for several hours, after which her ear seemed much cleared and the tinnitus ceased (release of lymphatic hypertension through the utriculosaccular valve?). The hearing was dramatically improved (see Fig. 16, lower). Unfortunately a few days later the roar-

Fig 7—Upper



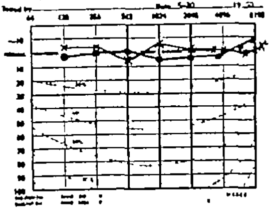
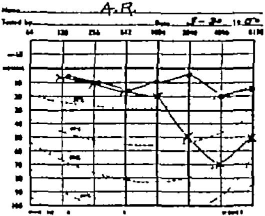


Fig 7—Lower

Fig 8—Upper



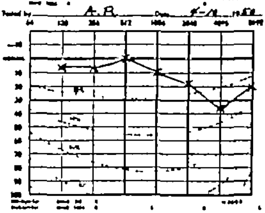
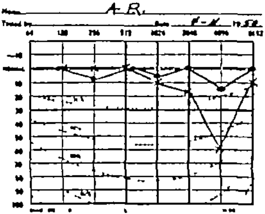


Fig 8—Lower

Fig 9—Upper



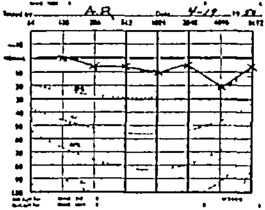
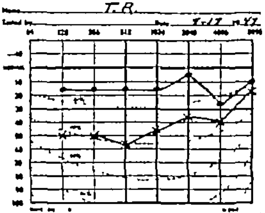


Fig 9—Lower

Fig 10—Upper



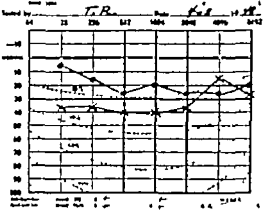
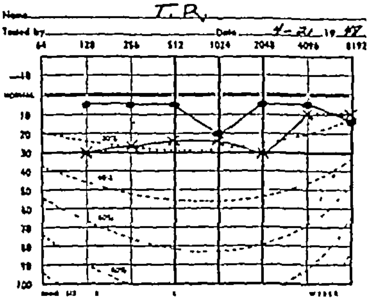


Fig 10—Lower

Fig. 11—Upper.



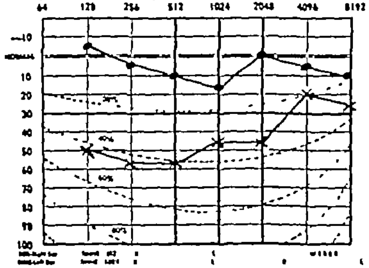
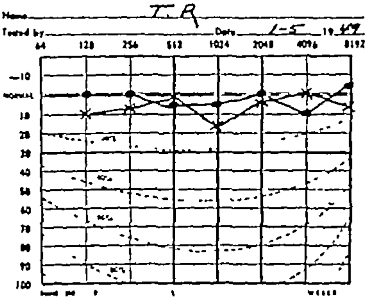


Fig. 11—Lower.

Fig. 12—Upper.



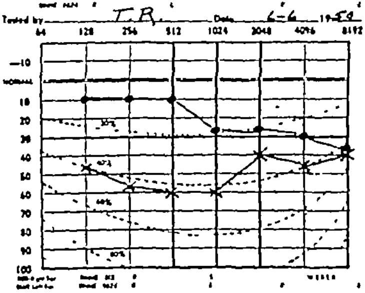
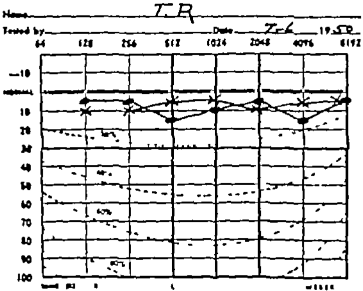


Fig. 12—Lower.

Fig. 13—Upper



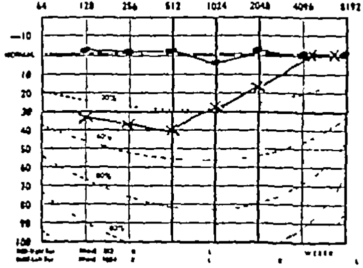
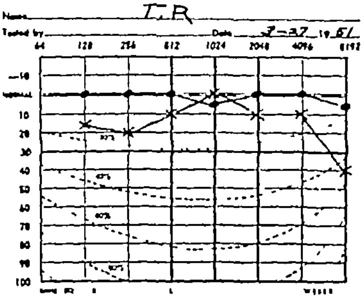


Fig 13—Lower.

Fig. 14—Upper.



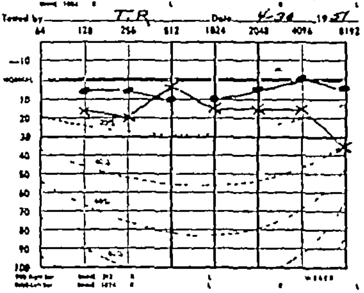
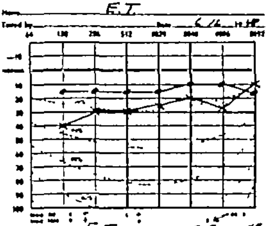


Fig. 14—Lower.

Fig 15—Upper



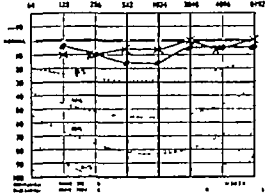
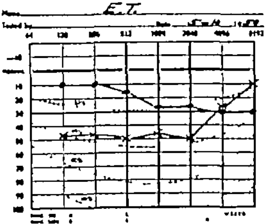


Fig 15—Lower

Fig 16—Upper



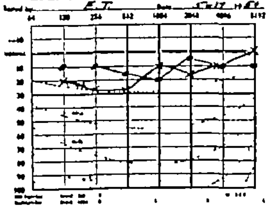
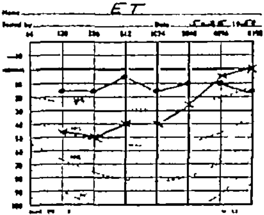


Fig 16—Lower

Fig 17—Upper



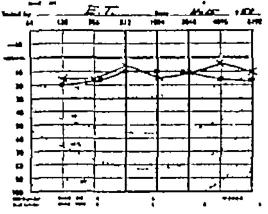
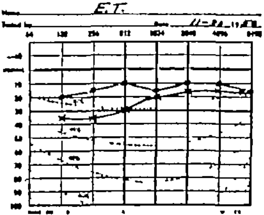


Fig. 17—Lower

Fig 18—Upper



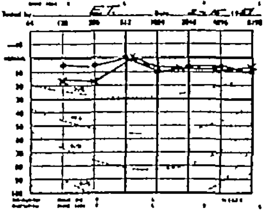
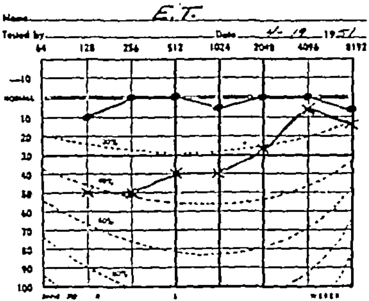


Fig. 18—Lower

Fig. 19—Upper.



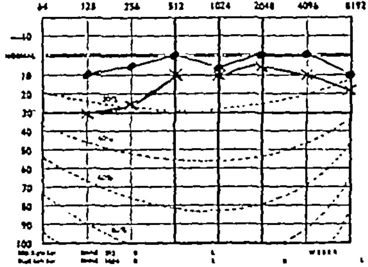
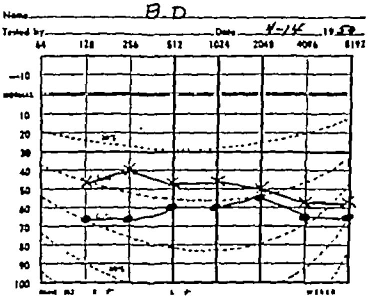


Fig. 19—Lower.

Fig. 20—Upper.



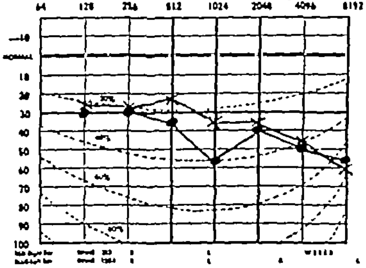
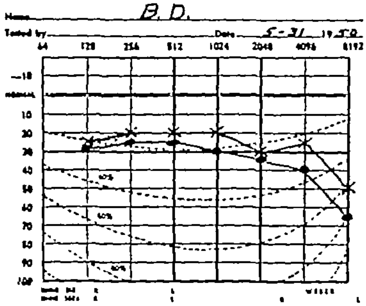


Fig. 20—Lower.

Fig. 21—Upper.



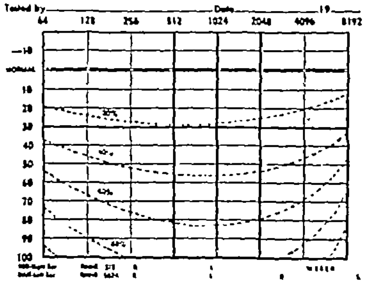
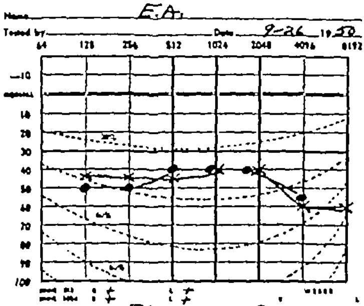


Fig. 21—Lower.

Fig. 22—Upper.



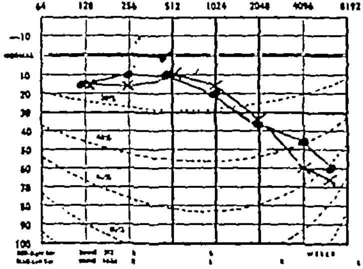
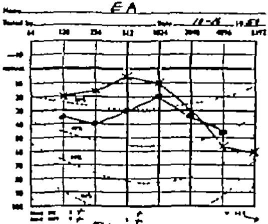


Fig. 22—Lower.

Fig 23—Upper



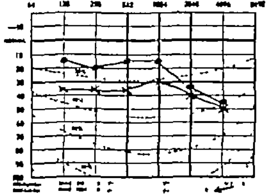
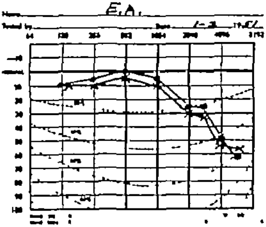


Fig 23—Lower

Fig 24—Upper



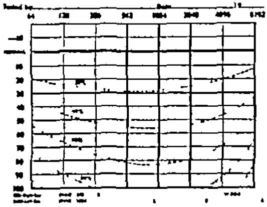
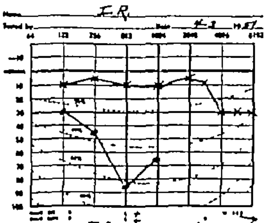


Fig 24—Lower

Fig 25—Upper



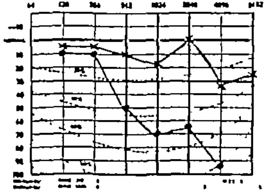
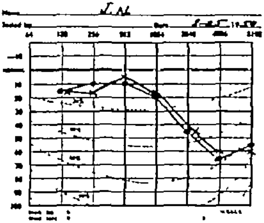


Fig. 25—Lower

Fig. 26—Upper



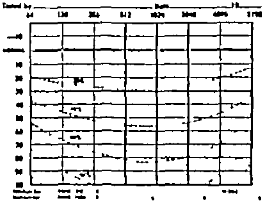
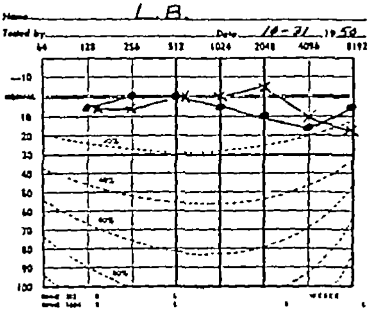


Fig 26—Lower

Fig. 27—Upper.



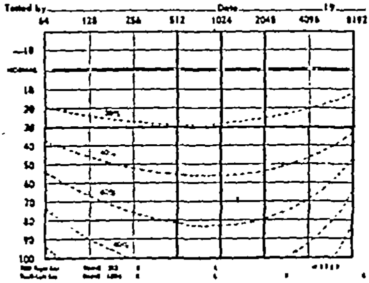
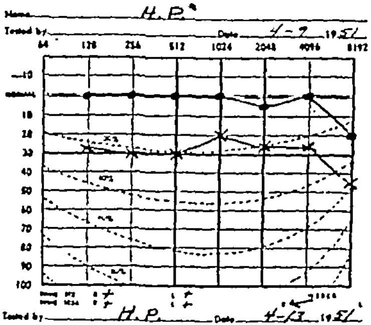


Fig. 27—Lower.

Fig. 28—Upper.



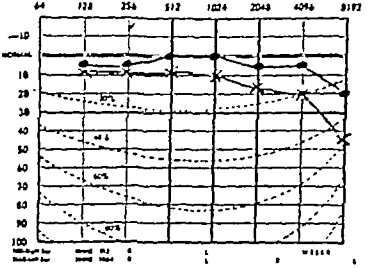
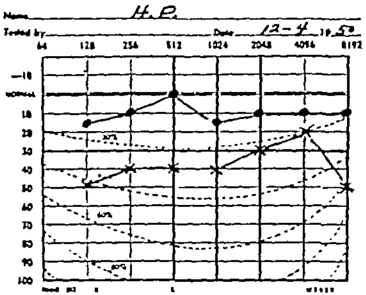


Fig. 28—Lower.

Fig. 29—Upper.



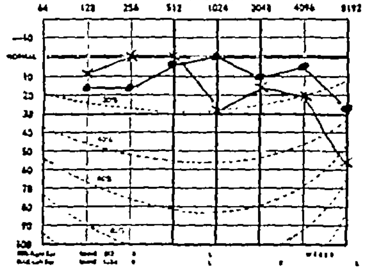
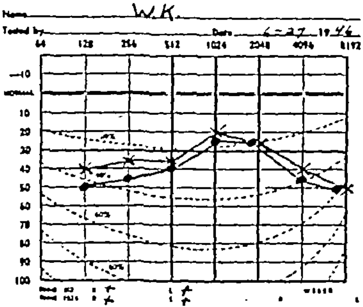


Fig. 29—Lower.

Fig. 30—Upper.



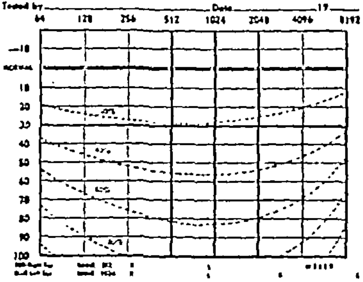
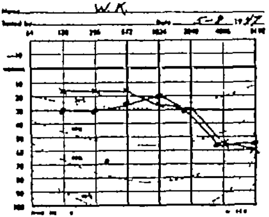


Fig. 30—Lower.

Fig 31—Upper



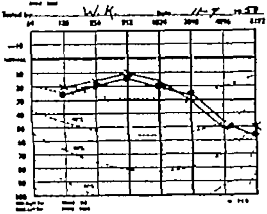
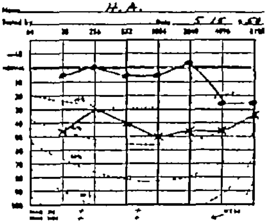


Fig 31—Lower

Fig 32—Upper



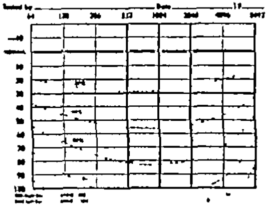
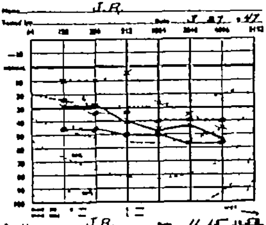


Fig 32—Lower

Fig 33—Upper



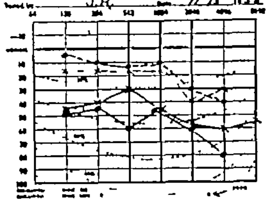
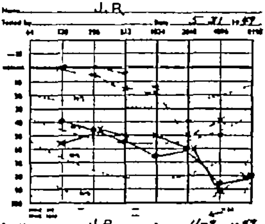


Fig 33—Lower

Fig. 34—Upper



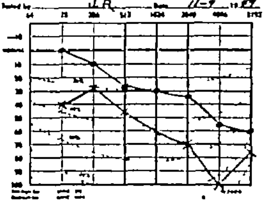
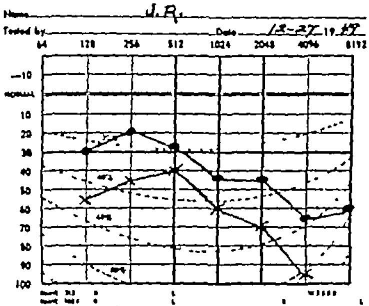


Fig 34—Lower

Fig. 35—Upper.



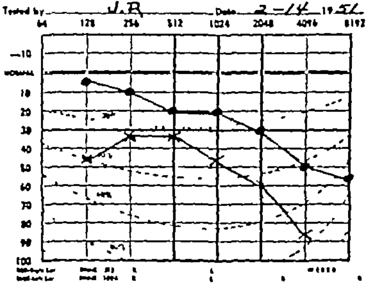


Fig. 35—Lower.

ing returned (see Fig. 17, upper). She improved during the Summer, but during the Fall ear symptoms developed again, only to clear by November (see Fig. 17, lower) after a severe prolonged period of mucous colitis with severe diarrhea. Whether the dehydration resulting from the dysentery or ACTH and cortisone, which were used at this time, were responsible for the excellent return of hearing was impossible to say. Two weeks later tinnitus and vertigo recurred, however (see Fig. 18, upper). By February the audiogram was again normal (see Fig. 18, lower) with a complete absence of tinnitus. Six weeks later the tinnitus, vertigo, and blocking again recurred. During the next few weeks the ear cleared somewhat, only to recur again with initial vertigo, aural pressure, and hearing impairment (see Fig. 19, upper). It has since gradually improved with treatment so that now only faint tinnitus is present (see Fig. 19, lower).

In virtually every exacerbation in this long and recurring course a period of emotional trial preceded recurrence — often accompanied also by an episode of mucous colitis. Her vertigo is usually only initial with each recurrence, as is usually the case in endo- or perilymphatic hypertension. Consequently, and in view of the potentially normal cochlea *present, therapeutic labyrinthine destruction would be a grievous error.*

The patient is being maintained on oral niacin 100 mg. four times a day one-half hour before meals and at bedtime (a mildly flushing dose for her), potassium chloride (Enseals) one-third gm. three times a day, diethylstilbestrol 0.1 mg. daily, and thyroid 60 mg. daily, plus vitamins B and C.

Comment: These cases are not exceptional in average otologic experience. They demonstrate the remarkable salvage in hearing which is possible when the lymphatic tension in *the labyrinth is relieved. It may recur and last for prolonged* periods of time, but a good prognosis for recovery is present if the high tones are essentially uninvolved.

Because the flat and rising audiometrical curves of lymphatic hypertension, together with the symptom of aural pressure superficially implicate the middle ear, these cases require extreme differential care. Accordingly we have in each instance had confirmatory examination and testing done independently by three competent otologists. In fact, Case 3 has been examined over a period of four years by six different otologists to confirm the inner ear source.

The next two cases represent a picture of lymphatic hypertension but have an uncorrectible high tone loss superimposed. Whether or not the high tone ganglionic loss in these cases resulted from ganglion cell ischemia occurring simultaneously with the lymphatic tension of stria vascular ischemia or independently as previous ganglion cell ischemia (as depicted in Cases 1 and 2) is impossible to say.

Case 5: B. D., female, age 69, when first seen on April 14, 1950, complained that for the past six months she had had a hearing problem which started in the right ear but later involved the left. It began with roaring and buzzing in the right ear, severe vertigo, nausea, and vomiting. Elsewhere she had been given a salt-free diet and nicotinic acid. Three months before examination the left ear had become similarly involved. She frequently experienced vertigo of mild degree associated with an increase in the ever present tinnitus and a disturbing feeling of fullness in the ears. In addition to hearing impairment, aural pressure and tonal distortion were extremely distressing. The audiogram showed marked loss of hearing (see Fig. 20, upper). The caloric response was normal on both sides and reduplicated the sensation she had had during her milder attacks of vertigo.

Intravenous procaine completely relieved the tinnitus on the first day it was used. After intravenous procaine on three successive days tonal distortion and aural pressure were relieved (see Fig. 20, lower). In the succeeding six weeks she had nine additional intravenous procaine injections and daily oral niacin, ascorbic acid, and B complex. The hearing improved (see Fig. 21) and there was no further pressure or vertigo.

She was advised of the possibility of exacerbation under the strain of her teaching program, and warned to lighten her load at first signs of recurrence and resume oral niacin and artane for a prophylactic three weeks under such circumstance.

Case 6: E. A., female, age 68, never had much difficulty with hearing until two days before examination. Then, during an anxious, nervous period she had a block develop in her ears with a feeling of pressure and marked distortion of sound plus a steady pulsing noise bilaterally (see Fig. 22, upper). She had also had a persistent sensation of vertigo of moderate degree for the two days. Examination revealed both middle ears to be clear. Blood pressure was 190/100. Her cranial nerves other than the VIIIth were intact. She was given 1 cc. of papaverine intramuscularly and oral niacin. The next day her hearing was markedly improved (see Fig. 22, lower). She has been continued on oral papaverine and niacin. She has occasional episodes alternating from one ear to the other (see Fig. 23, upper and lower) of tinnitus. aural pressure and mild vertigo which have been aborted with intravenous papaverine; however, her general course has been satisfactory, as shown in her most recent audiogram (see Fig. 24). This in spite of blood pressure elevation at times up to 260/140.

Case 7: This is a case of *total cochlear vasomotor ischemia* (see Fig. 5). As the case study will show, the prognosis for the return of function in such cases is poor. This is especially true if the hearing for the higher frequencies is *nil* when first seen following the vasomotor crisis.

I. R., male, age 53, was first seen on April 3, 1951, complaining of hearing loss and noise in his right ear (see Fig. 25, upper). Eleven days previously the patient had been at cemetery services where a wind blew against his unprotected right ear for a prolonged time. Thereafter he noticed his hearing drop within a matter of hours, and he developed a broad confused noise in the right ear. He had noticed no severe vertigo.

but had had a minor episode which made him careful of rapid movements. The sensation of fullness and pressure in his ear was distressing. General health had been good previously. He had been seen elsewhere on the day after onset and had been started on nicotinic acid and roniacol tartrate without improvement.

Examination revealed normal drum heads and middle ears. There was loud voice perception only inconstantly at contact in the right ear when the left was masked. The caloric responses were equal bilaterally. He was given intravenous procaine with 50 mg. of nicotinic acid per 250 cc. of diluent and was continued on oral niacin, roniacol tartrate and was given artane and vitamins B and C in addition.

By April 9, 1951 — after five intravenous procaine injections — he heard soft voice at farther than arm's length in the right ear. There was less pressure and tinnitus.

On April 12, 1951 — after nine intravenous procaine with nicamin injections — he heard voice (although not recognizable) over the telephone for the first time.

There was no further improvement with subsequent vasodilating therapy (see Fig. 25, lower). The patient is being maintained on oral nicotinic acid, vitamin B complex, and ascorbic acid because his nervousness and depression over the episode frequently excite a transient aural pressure and fine tinnitus in the opposite ear. He has many symptoms suggestive of the male climacteric and maintains much better general tone with 5 mg. of methyl testosterone sublingually twice a day.

Comment: This case points up the medically emergent character of labyrinthine ischemia. Ganglion cells deteriorate quickly in the presence of severe ischemia. Hours are important. Delay of days and weeks in correction of ischemia usually results in severe, irreversible disability.

The vestibular portion of the labyrinth as a whole or its individual segments can be involved by vasomotor ischemia.

Ischemic impairment of one ampulla is not uncommon (see Fig. 3).

Case 8: J. N., male, age 63, had had an abrupt attack of vertigo with nausea and vomiting two months before examination. He had experienced no fullness of the ear, tinnitus, or hearing loss. He had had no further spontaneous attacks since the initial one, but ever since had a most distressing sensation of vertigo when he moved his head in the anterior vertical plane, as when looking upward.

In the face of this persistence one must presume residual ischemic damage to the neuroepithelium of the crista of the anterior superior semicircular canal. It was of a degree that was symptomatic, yet not demonstrable by the crude inadequacy of the caloric test. His audiogram (see Fig. 26) showed a bilateral high tone hearing loss which was presumably old. He was placed on vasodilating therapy orally as a precaution against ischemic recurrence. So far as the defective signal from the damaged end-plate was concerned the patient was reassured that in due course the balances of compensation and suppression would rid him of the positional annoyance.

The utricular vascular supply may be involved singly (see Fig. 3). A strong sensation of pulsion results. If ischemia is severe enough to damage the macular end-plate such an affected individual maintains, during the compensatory period, a feeling of pitching forward while in motion as though being pushed.

Case 9: L. B., female, age 47, had the sensation of falling forward whenever she was in motion for several weeks following a sudden severe vestibular attack associated with nausea and vomiting with no apparent hearing loss (see Fig. 27) or tinnitus.

Comment: The problem in an instance of this sort is whether to allocate the lesion to the end-organ or to the central nuclear area, since there is no localizing cochlear symptom. The problem seems resolved, however, when one appreciates the extraordinarily unlikely fact of discrete plane involvement in the diffuse anatomy of the nucleus.

Where more than a single crista or macular plaque is involved in the vestibule the residual vertigo is more diffuse and not limited to production in only one place of motion. This, of course, is the more common picture.

Case 10: The possibility that the *utriculosaccular* valve described by Bast and Anson and others can suddenly release endolymph under pressure from the cochlear to the vestibular side of the labyrinth is illustrated by H. P., female, age 42, with a past history of fluctuating hearing in the left ear, who developed a marked hearing loss with no perception of loud voice which persisted for three days and which suddenly "cleared" markedly in a matter of hours coincident with a sudden attack of vertigo. Examination 24 hours after the vertigo showed what she regarded now as a comparatively "clear" left ear to be still significantly impaired (see Fig. 28, upper). Apparently the unmeasured impairment which had "cleared" to this level must have been originally very severe. Within a week on oral nicotinic acid, thyroid and diethylstilbestrol therapy there was excellent return (see Fig. 28, lower). This same patient, nine months before, had had an equally severe lymphatic tensive impairment (see Fig. 29, upper) persist for five months without treatment and then clear to normal hearing promptly after taking nicotinic acid, thyroid and diethylstilbestrol orally for a week at that time (see Fig. 29, lower).

Case 11: That prolonged vasodilator therapy can prove useful in *preventing gradual progressive hearing loss* and in regaining the recent increment of impairment in a vasomotor cochleitis is evidenced by W. K., male, age 60, who was first seen on June 27, 1946, complaining of progressive loss of hearing and bilateral tinnitus. He had had chronic vasomotor rhinitis and bronchitis for over 20 years, exhibiting marked sensitivity to interior dust and to physical change. During the four months preceding examination he and his intimates had noted a disturbing increase in the rate of decline of his hearing (see Fig 30) He has been taking oral nicotinic and vitamin B complex since that time. His tinnitus is now only occasional and his hearing has improved during the last five years (see Fig. 31, upper and lower)

Case 12 Vasospasm is not limited to the labyrinthine arterial bed. This is illustrated in a case with *simultaneous cerebral and labyrinthine vasospasm* H A., male, age 47, was first seen on May 15, 1950, with a complaint of recurrent attacks of vertigo and unconsciousness. These dated back two years. The first attack had left him hard of hearing in the left ear with constant tinnitus on that side. He was unconscious for two or three hours at that time. The attacks since had occurred as often as several times a month with severe vertigo the prominent symptom. Unconsciousness frequently but not always accompanied the vertigenous attacks and was associated with numbness of the right hand and leg Neurological examination had been negative. His audiogram showed a flat 50 db loss in the left ear (see Fig 32) Caloric testing showed a minimal residual response on the left with massive douching. The reaction on the right was somewhat delayed but in the main normal

History revealed that previous to the onset of his illness two years ago he had done well running a department store in South Dakota. At that time he sold out and bought another store in Minnesota. He then was caught in an effort at expansion in the building jam of the postwar period It was at this time that the ear and cerebral symptoms developed.

The patient's internist had first treated him aggressively with intra venous histamine and oral vasodilators and had then had neurological and neurosurgical consultation relative to an VIIIth nerve section. The neurosurgeon had suggested labyrinthotomy as a more satisfactory procedure.

It was felt that labyrinthotomy would be purposeful to control the vertigo and that there was little harm in destroying the slight cochlear reserve, but that it would hardly control the intracranial vasospasm. Accordingly he was urged to retire or unload his business pressures in some manner in an effort to control both his labyrinthine and his intracranial vasospasms, rather than have the labyrinthotomy done to answer only half his problem. He was given an oral maintenance dose of nicotinic acid, artane, and phenobarbital.

Since he complied with this advice he has had only two very minor momentary attacks of vertigo and no unconsciousness. His hearing level remains reduced to the same degree and the caloric response on the left remains virtually absent.

Vasomotor changes occur in the inner ear of the otosclerotic, too, and an understanding of this fact may aid in the selection of suitable fenestration candidates, and may explain some of the eventual failures of fenestration surgery.

Case 13 J R., female, age 42, is an *otosclerotic* who was judged unsuitable for *fenestration surgery* in 1946 because she had had recurrent attacks of vertigo and exacerbations of right ear tinnitus for two years Her first examination audiogram (see Fig 33, upper) showed the picture of otosclerosis with superimposed labyrinthine stria vascular ischemia (note the depression of the bone conduction on the right side) Pyribenzamine, a capsule combining amytal, scopolamine, and atropine, and intravenous histamine were used at various times during 1946 to control the vertigo. In May of 1947 severe headaches of several days' duration associated with increased roaring in the ears were immediately controlled by 3 cc. of tetraethylammonium chloride intravenously on two occasions. Extensive effort was made to have her curb her ambitions and perfectionisms and live within her emotional means. During 1948 she was comfortable save for mild seasonal pollenosis. She was also comfortable during 1949 and 1950 and an audiogram at that time (see Fig. 33, lower) revealed absence of the previously lowered bone conduction on the right.

Comment: In view of the absence of lymphatic hypertensive symptoms during the last three years, this patient might now be considered more seriously for fenestration surgery, although she does have a basic systemic vasomotor instability (headache, pollenosis, labyrinthine ischemia) which might at any time jeopardize the benefit resulting from the surgery.

Case 14: The postfenestration effects of a vasomotor reacting inner ear are shown by J. R., female, age 40. She was first seen on May 31, 1949, complaining of hearing loss in both ears of 12 years' duration. Tinnitus was present bilaterally. No vertigo was complained of. An audiogram (see Fig. 34, upper) showed markedly reduced hearing bilaterally. Bone conduction audiometry revealed the 2,048 frequency and those higher to be markedly depressed. Tuning fork tests confirmed the bone conduction audiometry. She was a borderline surgical case. A fenestration was performed on the right ear on June 22, 1949. Postoperatively her course was good and on Nov. 9, 1949, her hearing recorded an average improvement of 25 db in the operated ear (see Fig. 34, lower).

On Dec. 27, 1949, she complained of a sensation of pressure, sound distortion, and hearing impairment which had been present in her operated ear for 24 hours. An audiogram (see Fig. 35, upper) revealed the hearing in the operated (right) ear to have dropped off about 10 db from its previous level. The fenestrum was patent. The patient had had a nervous, anxious 48 hours preceding the onset. This period of anxiety had terminated and it was decided to give her natural corrective neurovascular processes opportunity to clear the ear.

One month later an audiogram revealed the original postoperative level regained; however, six weeks after this she was given oral nicotinic acid and B complex to be used during symptomatic periods because she had come to note that at the time of her menses or frequently at times when she was tired or nervously upset she would develop tinnitus and aural pressure in her operated ear. In an enthusiasm of overdoing, she used the medications most of the time. It controlled her symptoms well. Her aggravant periods were minimized. An audiogram (see Fig. 35, lower) one year later revealed not only improvement in the hearing in the operated ear, but also improvement in the hearing in the unoperated ear. It is probable that unsuspected vasomotor cochlear impairment had been present even before operation.

Comment: Otologists have frequently remarked improvement in the unoperated as well as in the fenestrated ear in certain otosclerotics following fenestration surgery. Some of *these may be cases of otosclerosis* in unstable vasomotor-reacting individuals who, when the emotional strain associated with hearing impairment has been lightened by a good fenestration result, regain a more normal general vasomotor tonus which in terms of the cochlea may mean improved hearing — even in the unoperated ear.

In otosclerotics with associated inner ear impairment with high tone drop we have made it a habit to check thoroughly into the neurovascular background and give due attention to the historical fluctuations of acuity associated with weather change, fatigue, menstrual-cycle, or emotional tension. Periodic dependence upon vasodilating and blocking agents, vitamin concentrations, and hormones where purposeful have paid *off in maintenance of our fenestration results.*

CONCLUSIONS.

1. The labyrinth is one of the most discrete and symptomatic reactors in the field of peripheral vascular disorder.
2. The dead end of therapeutic nihilism no longer applies to that large segment of inner ear disorders resulting from peripheral vasomotor change.
3. By aggressive neurovascular stabilization a significant segment of imperiled inner ear function can be salvaged and maintained.
4. A considerable responsibility rests with otologists to teach the early recognition of these vasomotor disturbances, so aggressive therapy can be applied promptly.
5. The otologist cannot be a narrow surgical technician. He has also an important medical mission to perform.
6. Otology in this regard has a fascinating and an unlimited horizon.

CONGENITAL TRACHEOESOPHAGEAL FISTULA NOT ASSOCIATED WITH ATRESIA OF THE ESOPHAGUS.*

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INTRODUCTION.

Congenital tracheoesophageal fistula is one of the less common developmental anomalies, but it has received considerable attention during the last 15 years and is now one of those anomalies which can be successfully treated by surgery. The increasing interest in the diagnosis of all congenital defects has resulted in the earlier recognition of an increasing number of cases of tracheoesophageal fistula *with* the usually associated atresia of the esophagus, and by this earlier recognition more successful treatment has been accomplished. Mortality in such cases is steadily decreasing, whereas up to 1939 no patient had survived surgery. In 1940, Lanman[1] reported 32 cases studied at The Children's Hospital in Boston between 1928 and 1938. All but one of these children had the usually associated atresia of the esophagus. Thirty of these patients were operated upon, with a mortality for the series of 100 per cent. During the following eight-year period (1939-1947) at the same institution, Swenson,[2] in 1948, reported 113 such cases, 57 of whom survived surgery—a mortality of only 50 per cent.

The purpose of this paper is to point out that there is now evidence that there is an increasing number of cases in which

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and blowing we found the wind had a vent, but not by the top of the gullet. Then we carefully slit up the backside of the gullet from the stomach upwards, and when we had gone a little above halfway toward the pharynx, we found it hollow no further. Then we began to slit it open from the pharynx downward, and it was hollow til within an inch of the other slit, and in the imperforate part it was narrower than in the hollowed. The isthmus (as it were) did not seem ever to have been hollow, for in the bottom of the upper and the top of the lower cavity there was not the least print of any such thing, but the parts were here as smooth as the bottom of an acorn cup. Then searching which way the wind had passed when we blew from the stomach upwards, we found an oval hole (half an inch long) on the fore side of the gullet opening into the aspera arteria (lower trachea), a little above its first division, just below the lower part of the isthmus above mentioned."

Thus there is no question but that over 250 years ago Gibson had made a most accurate observation of a congenital tracheoesophageal fistula with the commonest of associated anomalies (esophageal atresia), in which the fistulous tract connects the trachea and the lower esophageal segment (Type III b. of Vogt[8]).

In 1873, Lamb[9] is credited with having been the first in North America to report a case of tracheoesophageal fistula without other esophageal abnormalities. The baby died at the age of seven weeks. In 1884, Mackenzie,[6] going back to 1670, collected 62 cases of congenital atresia of the esophagus, 43 of which had a tracheoesophageal fistula, and added one of his own. In 1917, Cautley[10] merely listed from the literature six cases of tracheoesophageal fistula supposedly without atresia, and Lamb's case[9] was one of them. He did not discuss the cases. In a very complete review of the literature up to 1919, Plass[11] reported 136 cases of the anomaly, all but one of which were associated with an esophageal atresia, and that one was the case of Lamb noted above. Haight[4] has taken the time to verify these six cases listed by Cautley. In addition to that of

Lamb there were five others. Eppinger's case was that of a congenital tracheoesophageal fistula just above the bifurcation of the trachea and a normal esophagus. The second case, reported by Vrolik, was considered by Plass as having an associated atresia, but according to Eppinger had a narrow esophageal stricture below the fistula. The patient described independently by Pinard and Tarnier was also considered by Plass to have an atresia, but Haight's consultation with the original reference determined that he had a high tracheoesophageal fistula just below the glottis and no atresia of the esophagus. The cases of Richter and von der Water could not be verified by consultation with the original papers.

Vogt,[3] in 1929, offered the first attempt at classification of tracheoesophageal fistula and congenital atresia of the esophagus, but did not mention the possibility of tracheoesophageal fistula occurring without atresia.

Type I. Complete absence of the esophagus. (Very rare type.)

Type II. Atresia of the esophagus with separate upper and lower segments, each ending in a blind pouch.

Type III. Atresia of the esophagus with a tracheoesophageal fistula:

- *a.* Fistula occurring between the upper segment of the esophagus and the trachea.
- *b.* Fistula occurring between the lower segment of the esophagus and the trachea (Commonest type.)
- *c.* Fistula arising in both segments of the esophagus and connecting with the trachea.

Ladd[5] has added a Type IV (a variant of Type III c of Vogt) in which there is an atresia of the esophagus with a fistula which arises at the bifurcation of the trachea instead of above it and enters the lower segment of the esophagus. One of Lanman's[1] cases (listed as Type III c) showed an upper esophageal segment with a tiny tracheoesophageal fistula as its only outlet, and a lower segment which also opened into the trachea at the same level. In this case the only communication between the upper and lower esophageal segments was a detour via the trachea (Type V of Ladd). Holt, Haight, and Hodges[13] have suggested adding another classification to include the subject under discussion—namely, tracheoesophageal fistula *without* atresia (see Figs. 1 and 2). In 1929, Negus[11] reported the case of a 45-year-old man who died of carcinoma of the lung Postmortem examination showed a congenital opening running upward from the esophagus into the trachea. No symptoms had been noted which could have been attributed to the fistula. This was felt to be due to the fact that a small valve-like hood of mucous membrane was

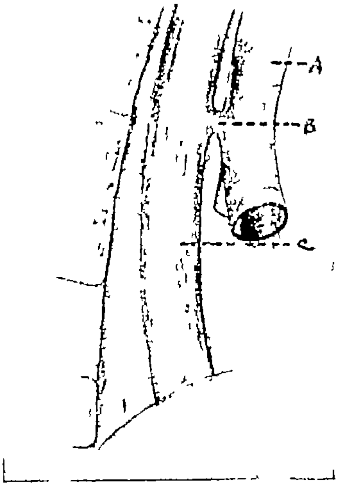

Fig 1 Schematic drawing of a tracheoesophageal fistula without atresia of the esophagus. (A) Trachea. (B) Fistula. (C) Esophagus. From Swenson (2) (p 199)

found on the esophageal aspect of the fistula. This flap may have temporarily closed the opening during swallowing and prevented the regurgitation of food into the trachea. In 1931, Rosenthal[14] in an excellent and comprehensive paper described both the gross and microscopic pathology found in cases of tracheoesophageal fistula. He reported 205 congenital tracheoesophageal fistulae occurring in a total of 255 cases of atresia of the esophagus, including eight of his own. Of this total group of 255 cases, 205 had the commonly associated fistula, 40 did not have a fistula, and 10 were not autopsied so its presence or absence was not ascertained. Rosenthal makes no mention of fistula without atresia of the esophagus. In 1933, McKinney[15] reported a patient whom he suspected had a con-

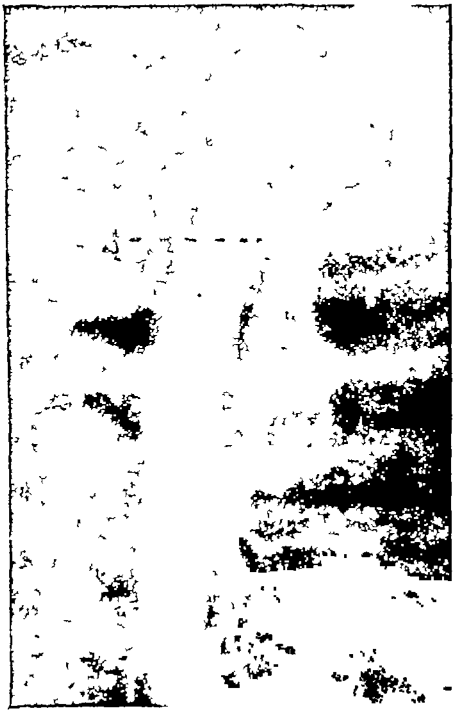

Fig 2 Oblique Roentgenogram of intratracheal injection of lipiodol showing tracheoesophageal fistula (A) with no atresia of the esophagus

genital tracheoesophageal fistula This was a 58-year-old woman who had for 25 years complained of gagging, vomiting, and a paroxysmal cough associated with and also following meals. A gastrointestinal series of Roentgenograms showed barium in the right bronchial tree. Although esophagoscopy was negative, bronchoscopy showed what was described as a "probable" opening in the tracheal wall from which a creamy fluid was emanating; however, this cannot be regarded as a proven case of congenital fistula.

It was not until 1939 that a successful cure of a case of tracheoesophageal fistula was first accomplished. In that year both Ladd and Leven successfully operated upon the first patients with congenital tracheoesophageal fistula associated *with* atresia of the esophagus. Leven[16] reported his case in 1941 Ladd's[8] case was 24 hours older but was not reported until 1944. In 1939, also, Imperatori[3] reported the first attempt to make a surgical closure of a congenital tracheoesophageal fistula *without* an associated atresia. This was an interesting case of a six and one-half-year-old boy who had been treated for years as a difficult feeding problem It was thought that he probably had a stricture of the esophagus so the esophagus was dilated several times Regurgitation of food continued, and the patient went through many bouts of bronchopneumonia While swallowing a string in preparation for one of the dilatations of the esophagus, the string suddenly went through a congenital fistula into the trachea and produced marked respiratory difficulty. Emergency tracheotomy was necessary. When the patient was six years old, Dr Chevalier L. Jackson demonstrated bronchoscopically a slit-like fistula 1 3 cm in length in the region of the third, fourth and fifth tracheal rings. Imperatori closed this fistula in two operations through a cervical tracheotomy incision and the patient thenceforth did most satisfactorily. In 1940, Lanman,[1] in a very thorough paper, reviewed the literature and presented 31 cases of fistula in 32 patients with esophageal atresia treated at The Children's Hospital in Boston. In 1941, Gengenbach and Dobos[17] stated that although tracheoesophageal fistula without atresia exists, such conditions are obviously quite rare. They did not mention Imperatori's case, but reported two patients of their own who were proven to have congenital tracheoesophageal fistula without atresia, and one in whom the possibility was suspected clinically but never proven. The first was a three months old baby girl who had had a history of repeated respiratory difficulty associated with attacks of cyanosis since birth. She had regurgitated many of her feedings and cyanosis was associated with feeding. Barium studies showed no esophageal obstruction, but the barium entered the trachea. This was suspected to be due to a faulty deglutition mechanism of the epiglottis. Following the ingestion of the barium mixture the baby developed a severe bronchopneumonia and died. Autopsy, however, showed a small tracheoesophageal fistula 4 mm. in diameter located 3 mm. above the bifurcation of the trachea.

The second patient was a ten months old baby girl who since the third day of life had experienced attacks of cyanosis and coughing while feeding. She had been cyanotic at birth and also had had repeated attacks of bronchopneumonia, Bronchoscopy by Dr. Holinger in Chicago showed a small orifice 2 to 3 cm. above the bifurcation of the trachea, through which clear frothy fluid exuded. An apparent esophageal obstruction was encountered 19 cm. from the teeth. There is no record of therapy in this case, and since she was still living at the age of two years she obviously did not have esophageal atresia.

The third patient was a two and one-half months old female child who had the same type of history with a persistent cough since three weeks of age, when she began to have choking and cyanotic attacks associated with feedings. Esophageal lipiodol injection was performed, following which fluoroscopic and film studies were made. On the films lipiodol was seen in the left main bronchus and trachea. The diagnosis was made only on this evidence, as no definite fistula could be demonstrated. Neither esophagoscopy nor bronchoscopy was permitted, and a fistula was merely suspected.

In 1945, Leven and Lannin,[16] in a discussion of 41 cases from the University of Minnesota Hospitals, mentioned two patients who had a tracheoesophageal fistula without atresia. Three cases in this series had a simple atresia without fistula, and the other 36 the usual combination of fistula with atresia. They state, however, that diagnosis before autopsy is extremely difficult because the fistula may be small, symptoms inconstant and the diagnosis open to question.

Although nearly 100 patients with esophageal atresia had been seen at The Children's Hospital in Boston up to 1946, none had been diagnosed as a congenital fistula unassociated with an atresia of the esophagus.

The fifth case in this present series is an autopsy report of that first patient with a fistula without atresia.

In 1946, Holt, Haight and Hodges,[12] in a series of 46 cases from the University Hospital in Ann Arbor, Mich., mentioned one case of congenital tracheoesophageal fistula without atresia. In 1948, Haight[4] reported the second case which he had seen at the University Hospital and is now credited with having reported the second successful operation on a congenital fistula without atresia. The only two cases reported in which the anomaly was recognized by endoscopic methods were those of Imperatori[3] (bronchoscoped by Jackson) and Gengenbach and Dobos[17] (examined by Holinger).

INCIDENCE.

In a discussion on the incidence of tracheoesophageal fistula without atresia it is impossible to obtain accurate information because of the paucity of cases reported. In the past it was nearly always considered that the fistula occurred in conjunction with an atresia of the esophagus. Many cases of fistula with an otherwise normal esophagus must have been missed, and the incidence, therefore, may be much higher than one would suspect even at the present time. With more widespread knowledge that such anomalies do occur, and with better diagnostic methods employed in patients with vague gastrointestinal and respiratory complaints, the incidence may be found to be even higher in the future. For the present, however, the incidence will have to be considred on the basis of cases with associated esophageal atresia. In 1948, Haight[4] stated that during the preceding 12 years 63 infants with congenital atresia of the esophagus had been seen at the University of Michigan Hospital and that of these, only two had a fistula in the presence of an otherwise normal esophagus.

In an excellent article on incidence and possible etiology of this condition, Ingalls and Prindle[19] reported a study of 107 cases of infants with esophageal atresia, 102 of whom had an associated tracheoesophageal fistula. All of these patients were seen at The Children's Hospital in Boston from 1936 to 1948 or were born at the Boston-Lying-In Hospital between 1934 and 1945. They found that six cases had occurred at the Boston Lying-In Hospital during this period where 30,497 live births were reported (one was stillborn). On this basis they estimated the frequency of tracheoesophageal fistula in the Boston area as at least 0.2/1,000 live births, the major variable being the missed case (stillborn) and asphyxial death in which no autopsy is performed. They quote Murphy, who reported, in 1947, 13 cases in 166,451 live births in Philadelphia in a five-year period—an incidence of less than one in 10,000. A study of the literature revealed no case of true tracheoesophageal fistula which recurred as a familial trait in successive generations. Mackenzie,[6] however, reported the condition in three children of the same generation fathered by one man but born of different mothers. Lanman's[1] series of 32 patients included in the same family two children with tracheoesophageal fistula and esophageal atresia (Type III b of Vogt[8]). One was a boy born in 1932 and the second a girl born the following year. In 1939, Grieve and McDermott[20] reported two cases in successive male births in the same family in 1937 and 1938. Both of these babies died and postmortem examinations were performed. The fistula entered the lower esophageal segment (Type III b). An interesting feature is that the mother and father were third cousins. Ingalls and Prindle[19] state that in their series the condition occurred more often in males than in females, with a ratio of 62:45;

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In an excellent article on incidence and possible etiology of this condition, Ingalls and Prindle[10] reported a study of 107 cases of infants with esophageal atresia, 102 of whom had an associated tracheoesophageal fistula. All of these patients were seen at The Children's Hospital in Boston from 1936 to 1948 or were born at the Boston-Lying-In Hospital between 1934 and 1945. They found that six cases had occurred at the Boston Lying-In Hospital during this period where 30,497 live births were reported (one was stillborn). On this basis they estimated the frequency of tracheoesophageal fistula in the Boston area as at least 0.2/1,000 live births, the major variable being the missed case (stillborn) and asphyxial death in which no autopsy is performed. They quote Murphy, who reported, in 1947, 13 cases in 166,451 live births in Philadelphia in a five-year period—an incidence of less than one in 10,000. A study of the literature revealed no case of true tracheoesophageal fistula which recurred as a familial trait in successive generations. Mackenzie,[6] however, reported the condition in three children of the same generation fathered by one man but born of different mothers. Lanman's[1] series of 32 patients included in the same family two children with tracheoesophageal fistula and esophageal atresia (Type III b of Vogt[8]). One was a boy born in 1932 and the second a girl born the following year. In 1939, Grieve and McDermott[20] reported two cases in successive male births in the same family in 1937 and 1938. Both of these babies died and postmortem examinations were performed. The fistula entered the lower esophageal segment (Type III b). An interesting feature is that the mother and father were third cousins. Ingalls and Prindle[10] state that in their series the condition occurred more often in males than in females, with a ratio of 62:45;

In a discussion of possible causes for such maldevelopment, Rosenthal[14] refutes all theories except that of altered cell growth along the lateral ridges. This defective or absent growth of the septa would result in a fistula, whereas deficient growth of entodermal cells in the dorsal wall of the foregut would result in atresia of the esophagus.

It had been considered by Kreuter[25] that esophageal atresia was the result of cessation or retardation of growth of the esophagus at a stage when the tube had become a solid bar of tissue with no lumen. This condition supposedly is produced by proliferation and concrescence of the epithelial lining of the esophageal tube. Later on, in the normal course of events theoretically the lumen is re-established by vacuolization followed by coalescence of the vacuoles; however, Rosenthal considers this theory untenable because Fōrssner[26] demonstrated that the "solid bar" stage does not appear until the 20 mm. embryo, yet the tracheoesophageal anlage is well formed and the trachea is completely separated from the esophagus normally by the 4 to 6 mm. stage. The finding of the anomaly in these young embryos under 18 mm. (described by Ysander, Gruenwald and Minot) is concrete evidence for the refutation of this theory. In adddition there is apparently some question as to whether the above described process of obliteration of the lumen into the "solid bar" stage with eventual vacuolization and re-establishment of the lumen occurs in the process of normal development of the embryo. Schridde[27] found in an extensive study of 50 human embryos a normal lumen in the esophagus at all stages of development.

Keith and Spicer,[28] in a report of three cases of malformation of the tracheoesophageal septum with atresia and fistula noted the association of a deep origin of the right subclavian artery crossing from the aorta to the right side between the two esophageal segments. Such findings give credence to the theory that abnormal pressure of such vessels or of an abnormally developing heart might result in the separation of the continunity of the esophagus. Rosenthal,[11] however, believes that such cardiac or vascular anomalies are the effects rather two completely separate hollow tubes, — the anterior trachea and the posterior esophagus. On the ventral surface of the newly formed trachea develop the lung buds. These changes occur during the second fetal month so that at the end of seven or eight weeks of intrauterine life the trachea and esophagus should have been completely separated and normally developed. By the time the embryo has reached the 11 mm. state it should either have a normal trachea and esophagus, or the anomaly under discussion will have been established. The cause of such an anomaly as a persistence of an opening from one of these tubes to the other (*i.e.*, tracheoesophageal fistula) or a complete obliteration of the lumen of the posterior tube (*i.e.*, esophageal atresia), is not clear. The defect is usually very constant and must be present very early in embryonic life. An arrest in development of the septum could produce an incomplete separation of the two tubes and result in a fistula. Cause for this arrest in one particular area, however, is the crux of the problem, but the answer is by no means clear. This arrest has more generally been regarded as the result of a primary deficiency in the developmental capacity of the entodermal cells in the lateral masses rather than the result of some extraneous factor. The occurrence of the same defect in both halves of Ysander's[22] monster embryo would tend to substaniate this thesis. In 1924, Ysander described an 8 mm. monster embryo of the type Thoracopagus tetrabranchius in which there was but one stomach and one heart anlage while the esophagus and respiratory apparatus were duplicated identically. Each "embryo" presented a typical tracheoesophageal fistula with associated atresia such as is seen in full-term babies. This is the youngest embryo showing such an anomaly. Gruenwald,[23] in 1940, reported the condition in a 9 mm. embryo of about the same relative age. Minot[24] reported an 18.1 mm. embryo in the Harvard Embryological Collection which had a similar deformity. The association of other congenital malformations, especially those of the gastrointestinal tract such as the commonly associated imperforate anus, or other atresias, makes one strongly consider an inherent defect of cellular development.

subplacental fluid within the chorion. By connection with the pericardial cavity the contents of the recesses might be subject to pressure changes as the heart filled and emptied. Compression of the foregut between the recesses could divide the esophagus along their dorsal borders and produce the characteristic anomaly under discussion. They change in form rapidly as the left one disappears and the right gives rise to the superior recess of the omental bursa. In an older embryo which still measured only 3 mm., Broman has shown the anterior ends of these recesses crossing the esophagus and yet already below the site of the anomaly. An embryo of 4 mm. figured as a whole by Bremer who clearly reconstructed most of its organs was used to show how the associated atresia and fistula presumably arise. Malformation of the pneumatoenteric recesses perhaps with undue pressure from their contents in a brief period toward the close of the first month could produce the anomaly. At 8 mm. it should be well established."

Dr. George L. Streeter, quoted by Rosenthal[14] in a personal communication, suggested the possibility that tracheoesophageal fistula was produced in the same manner as spina bifida and presented this simple analogy. In the stage when the neural tube is open, closure takes place not by mesenchyme pushing in the entoderm, but by cellular proliferation which later results in fusion. If there is a deficiency in the cells resulting in altered proliferation and nonunion, the tube remains open (Spina bifida). In the tracheoesophageal situation the two tubes, a dorsal esophagus and ventral trachea, normally are formed from one original tube as has been outlined. If for any reason a deficiency in the cells which should form the septum causes altered proliferation and nonunion, a tracheoesophageal fistula persists. The deficiency in the dorsal entodermal cells that were to give rise to the esophagus directly above this area may result in its nonformation (*i.e.,* atresia). In the very early embryo (presomite stage) the anterior termination of the neurenteric canal is in direct proximity to that portion of entoderm where defective growth occurs. There may, therefore, be some correlation between than the causes of the development of esophageal atresia. The esophageal defect is already present, so during the process of development of the vascular tree these early vessels come to lie in the most easily accessible positions—namely, in hollow spaces in which there are no other organs, as in the area between the two pouches of the separated esophagus. In Cases 3 and 4 of the present series it is quite interesting that on fluoroscopic examination by lipiodol swallow a posterior esophageal defect was seen running obliquely upward from left to right at the level of the second or third thoracic vertebrae. This was interpreted as the characteristic appearance of an aberrant subclavian artery. At the time of operation in Case 3 this artery was readily identified as such, and was ligated and divided before the fistula was closed. Case 4 of the present series also presented the Roentgenologic appearance of an aberrant right subclavian artery. Gengenbach and Dobos[17] also suggested that the most plausible theory for this abnormal development was one advocated by Zausch (*Arch. f. Path. Anat.*, 239:94:1921), that pressure on the primitive entodermal sac by the enlarging heart anlage was sufficient to cause the anomaly. Rosenthal,[14] however, states that such pressure is greater on the trachea than on the esophagus, yet it is the latter structure which shows atresia. He also feels that there is an adequate space between the developing heart and tracheoesophageal anlage. In the paper by Ingalls and Prindle,[10] Dr. Frederick T. Lewis, of the Harvard Medical School, discusses a possible explanation along the lines of abnormal pressure in the developing young embryo. He quotes a monograph by Broman on the development of the omental bursa:

> "In human embryos of 3.4 mm. the common cleft-like cavity that later becomes vertically subdivided into the ventral trachea and the dorsal esophagus is closely flanked on either side by similar extensions of the peritoneal cavities. These extensions Broman named significantly the right and left 'pneumatoenteric recesses' but without recognizing their possible relation to fistula and atresia. Any fluid that they contain would be separated from that within the amnion and within the yolk-sac, but at that stage it would not be separated from

and certain commonly associated defects of the cardiovascular, gastrointestinal, genitourinary, respiratory and skeletal systems may represent departures from normal sequences of development at about the fifth or sixth week of embryonic life caused by agents acting through the mother and the placenta. Significant is the association of 20 cases of hydramnios occurring in the total group of 87 mothers of infants with such malformations of the esophagus. In this group four mothers had twins. In six instances antepartum hemorrhage occurred (five of these were during the first or second months of pregnancy). Six of the mothers suffered from acute infections or metabolic disturbances in the first trimester of pregnancy. These conditions were listed as acute upper respiratory infections, pyelitis, appendicitis and pneumonia. A possible metabolic factor in one patient was the ingestion of 30 gm. of quinine for an attempted abortion.

Ballantyne,[21] Brigham,[30] Meyer[31] and Scheurer[32] noted an increased incidence of hydramnios associated with these anomalies and believed it to be the result of the inability of the fetus to swallow and absorb amniotic fluid. They considered the usually noted dilatation and hypertrophy of the upper esophageal pouch in cases of atresia the result of ineffectual attempts on the part of the fetus to swallow this fluid.

Strong and Cummins[33] give credence to the theory of both genetic and environmental factors by stating that the agents predisposing to this anomaly are to be sought either in the reproductive cells from which the child is produced, or in an unfavorable intrauterine environment existing during the critical early period of embryonic life.

In a discussion of arteriovenous fistula, Reinhoff[34] states that there is in the life cycle of a cell or tissue which is differentiating or growing, a period in which that cell is very sensitive to its environment, and any injury to it at that stage will result in the abnormal growth and development of that cell or structure.

The problem, therefore, still exists and, so far there has been nothing but speculation as to possible causes of this, as the establishment of spina bifida and tracheoesophageal fistula. Mackenzie's[6] story of three children of the same father but of different mothers very strongly suggests an inherent genetic factor which may be responsible for the altered cellular growth in the local area described above.

Trauma and intrauterine inflammation or infection have always been propounded as possible causes of this as well as of many other congenital anomalies (*e.g.*, Luschka and Klebs, cited by Rosenthal[14]). In a paper in 1947 on epidemiological implications of developmental arrest, Ingalls and Gordon[20] discussed agents which could be regarded as possible causes of prenatal disease. They divided such agents into three categories: namely, *1. Infectious* (rubella, syphilis, etc.); *2. Chemical* (nutritional deficiencies, toxic and metabolic disturbances, etc.); and *3. Physical* (mechanical trauma, structural disease of the uterus, etc.). It is now generally accepted that an infectious agent may produce changes in the developing embryo of humans, as has been well known for some time in other animals, and that often these changes have a constant pattern from animal to animal. During the past several years since the studies of an epidemic of rubella in Australia it has become common knowledge that such an infectious agent as this particular virus when contracted by a mother in her first trimester of pregnancy very often produces permanent structural changes in the developing embryo. These changes are in the nature of congenital malformations which run a rather constant pattern: namely, the association of congenital cataracts; total deafness on the basis of VIIIth nerve or cochlear malformations; microcephaly; and various cardiac defects. All of these organs affected are in the process of maximum embryonic development during the first trimester of pregnancy. Mothers contracting rubella after the first trimester of pregnancy do not tend to produce infants with such defects. In Mongolism, Ingalls has postulated that the specific clinical defect produced is a nonspecific function of the stage of embryonic development, and not a specific property of the individual agent. Ingalls and Prindle,[19] therefore, conclude that it is possible that congenital tracheoesophageal fistula have been found. Rosenthal[14] quotes the work of Ladwig, who reported a case in which the lower portion of the esophagus continued into the posterior tracheal wall, and thick circular and longitudinal muscle layers and esophageal glands were found in the trachea itself. In the posterior tracheal wall stratified squamous epithelium extended up as far as the thyroid cartilage. Ladwig considered the posterior tracheal wall in such cases to be in reality an esophageal structure rather than a tracheal structure. Konopacki (from Rosenthal[14]) also found squamous epithelial cells in the tracheal mucosa in the region of the fistula and an abnormally thick muscular layer of the trachea where there were many cross-striated fibres. These findings were also corroborated by Gutmann[14] who noted that the muscle fibres of the posterior esophageal wall continued into the anterior tracheal wall as well. Rosenthal was the first to note tracheal structures in the lower portion of the esophagus. In three of his four cases studied histologically, the mucosa of the lower esophagus near the fistula was of the tracheal type with pseudostratified columnar epithelium, and in all four cases glands were found scattered through the muscle fibres of the esophagus. Respiratory epithelium in the esophagus, however, has been reported before in the case of normal newborn infants.[25]

The location and size of the fistula, as well as the adherence of the trachea and esophagus, have a bearing upon the surgical treatment of the fistula and upon the difficulties expected to be encountered.

SYMPTOMS AND DIAGNOSIS.

With such marked improvement in the surgical techniques which have brought about a steadily decreasing mortality in cases of congenital tracheoesophageal fistula or atresia of the esophagus, such cases must be diagnosed early so that proper surgical treatment can be instituted. In addition, it must be borne in mind that congenital tracheaesophageal fistula does exist without an associated esophageal atresia, and that in such cases the symptomatology may not be as striking as in the usually encountered anomaly which has an associated atresia. In the presence of vague respiratory and digestive complaints this condition must be suspected until the possibility of its existence is adequately eliminated. Often there may be merely a history of several admissions to the hospital for recurring bouts of acute pneumonitis or bronchopneumonia. Perhaps the story may be even less striking and the patient is considered simply to be unusually susceptible to otherwise insignificant respiratory infections. In many patients the persistence of a loose chronic cough may result in the diagnosis of chronic bronchitis and no further attempts are made to evaluate the situation. With greatly increased use of bronchoscopy and lipiodol studies in infants, many cases of recurrent bronchopneumonia are now found to be due to congenital anomalies of the tracheobronchial tree, such as stenosis of a bronchus, abnormal origin or abnormal twisting of a bronchial segment, or partial agenesis of the tracheobronchial tree or pulmonary system. Reliance in the simple elimination of a stenosis or atresia of the esophagus is dangerous, as the patient still may have a fistula.

In many cases of congenital tracheoesophageal fistula, if the opening is sufficiently small, the baby may be able to nurse properly and take his feedings satisfactorily. He may show a steady weight gain with no abnormal symptoms. In other cases, attacks of choking while swallowing may occur with or without cyanosis. Very often such patients are able to take solid or semisolid food with much more ease than liquids. In such patients the possibility of a congenital tracheoesophageal fistula should be strongly considered. Attacks of choking or cyanosis may occur during a feeding or sometimes at a considerable time after feeding, especially if the infant is placed in a prone position as so often is customary. In such a position he tends to regurgitate feedings which by gravity pass anteriorly through the fistula into the tracheobronchial tree. An increased amount of air in the stomach and intestines may lead to suspicion of a tracheoesophageal fistula. Inasmuch as infants normally have a considerable amount of air in the gastrointestinal tract as most of this air is swallowed during feeding, it becomes difficult to decide whether the amount is or is not abnormal; however, in cases of fistula, by closing the epiglottis as occurs during crying, vomiting, or straining, pressure in the tracheobronchial tree is thereby increased and air rapidly passes posteriorly through the communication into the esophagus and thence dilates the stomach. The cricopharyngeus muscle must also close spontaneously at this time so that the air is not passed to the outside through the pharynx and mouth, but is forced downward into the stomach and intestine, with resultant marked distention.

There is generally an increased amount of mucus in the mouth, pharynx and nasopharynx of such an infant, and very often the mucus may have a frothy appearance. A persistent loose, rattling type of cough occurs, and vomiting while coughing is frequently noted. The mucus, however, is not so troublesome as in patients with associated atresia of the esophagus, where it is necessary to employ constant suction to prevent further aspiration into the tracheobronchial tree.

Clinical examination of the patient may show good nutrition and other wise normal development, as most of these babies appear normal at birth, and can maintain adequate growth if the repeated respiratory infections are properly *controlled. This is much easier today* with the *extensive use* of chemotherapy and antibiotics. Often, however, other serious congenital defects will manifest themselves. The chest usually shows coarse rales and rhonchi throughout both lung fields, but very often these signs are more localized to the right upper lobe. Examination for tactile fremitus indicates an increased amount of mucus in the entire pulmonary system. The abdomen may be distended and tympanitic especially after crying, straining or even vomiting.

In the presence of choking and cyanosis during feedings, passage of a catheter from the mouth to the stomach should be the first diagnostic procedure in the case of a newborn infant. If an atresia is found, the obstruction is usually met when the catheter is 10 to 12 cm. from the alveolar ridge and its position with the fluoroscope is seen at the level of the second thoracic vertebra. If, however, no difficulty is encountered

Once a fistula is suspected, examination is not complete until bronchoscopic and esophagoscopic studies have been performed. These procedures should be used much more commonly in the infant age group than is generally the case. There

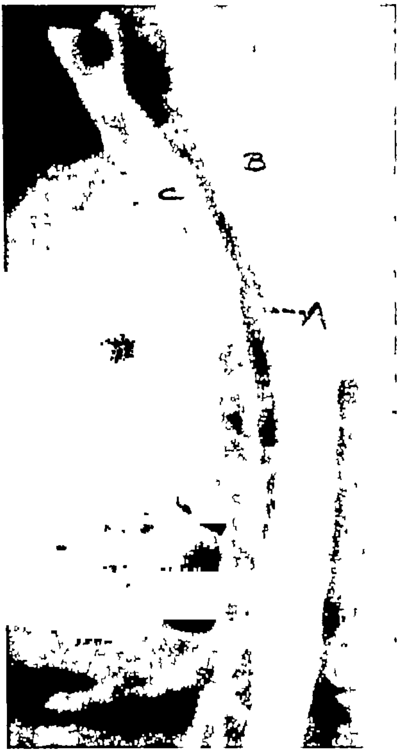

Fig 3 Lateral lipiodol Roentgenogram showing "tent-like" defect on anterior esophageal wall (A) at site of fistula. (B) Esophagus (C) Trachea. (From Swenson (2) (p 109)

is no contraindication to the use of these methods even in newborn infants, and such procedures at this age are very frequently necessary. It is, to be sure, a somewhat more difficult problem when it is necessary to use a 3 or 3.5 mm. bronchoscope and a small fistula may be very difficult to visualize through such a narrow lumen. The use of a Broyles telescopic bronchoscope simplifies the problem as direct visualization of the fistula can thereby be accomplished. More careful inspection of the lower posterior tracheal wall in the region from 1.5 cm. above the bifurcation downwards should be performed as most of the fistulae occur in this locality. In the demonstration of traumatic fistula in adults, Abbott[30] has found that when the patient has been given a swallow of methylene blue prior to bronchoscopy the fistulous tract may be more easily seen by tracheoscopic examination as the dye leaks through the communication. Such a solution can be injected through an esophageal catheter during the bronchoscopic procedure in an infant, and visualization of the fistula will be much easier. It is preferable to shift the baby carefully into the prone position while this is being done so that dye will run through the fistula by gravity. Esophagoscopy should be carried out in undiagnosed yet suspected cases, although bronchoscopic examination is much more apt to result in better visualization of the fistulous tract as the tracheal lumen does not undergo such wide or constant movements as the esophagus, and the tracheal rings add to the stability of the wall. In Haight's[4] case of the four-year-old boy, examination with the 5 mm. esophagoscope failed to show the fistula. It was also impossible to see the opening from the tracheal side through a 4 mm. bronchoscope. When a 5 mm. bronchoscope was employed, a large fistula was identified although the site of the fistula had not been localized by fluoroscopic examination with lipiodol injection nor by subsequent study of the films. The oil, however, had been seen to enter the trachea on the first swallow, so the diagnosis had been strongly suspected.

A technique which I have previously used to outline a fistulous tract between the esophagus and a bronchus is of service in the diagnosis of a congenital tracheoesophageal fistula not apparent by routine methods. A thin latex rubber balloon attached to a small, tightly ligated catheter is passed through an esophagoscope into the lower portion of the esophagus. The balloon is then inflated to close the esophageal lumen. Another similar balloon, which in addition has a second catheter passing either through or along the outside of the balloon (so that it projects just beyond the balloon's distal limit) is inserted into the very upper portion of the esophagus. The central portion of the esophagus can thereby be distended by the injection of air through the second or uppermost catheter into the closed esophageal segment. A small amount of lipiodol and air is then injected through the catheter and the increased pressure forces the lipiodol into the trachea via the fistula. Very little pressure is necessary. The fistula can be well outlined, and the troublesome question of reflux up the esophagus and aspiration of lipiodol through the glottis is eliminated. This is of considerable value in the sometimes troublesome differential diagnosis of a faulty neuromuscular mechanism of swallowing with regurgitation of lipiodol and subsequent glottic aspiration. It also has the advantage of outlining a small fistula which might not be apparent when lipiodol merely trickles down the lumen of the esophagus under no pressure.

TREATMENT.

Before proper operative treatment can be instituted, it is obvious that a diagnosis of fistula must be made, or at least that the presence of a fistula must be very strongly suspected. It is most helpful, however, to be able to localize the fistula. Early diagnosis is important before the inevitable and irreversible changes in the pulmonary system have been produced by repeated attacks of tracheal aspiration. Many of the above methods of diagnosis must be repeated once or possibly twice or even three times before the presence of a congenital tracheoesophageal fistula is satisfactorily eliminated.

When the diagnosis has once been made, ligation and division of the fistula by a direct surgical approach offers the only hope of satisfactory cure. It has been found that chronic or recurrent pneumonitis is an indication for early operation rather than a contraindication to it. A pulmonary infection will not subside until the communication between the trachea and esophagus has been tightly closed. Gavage feedings should be instituted as soon as the diagnosis is made and employed through as short a period of waiting as possible prior to operation. As soon as the baby's condition permits, thoracotomy should be performed.

In cases of traumatic perforation of the esophagus causing a tracheoesophageal fistula, Clerf and his associates[37,38] have used a bead of silver nitrate or crystals of sodium hydroxide applied by esophagoscopic methods to cauterize the borders of the fistula. These methods, however, do not seem appropriate or feasible in a case of congenital tracheoesophageal fistula in an infant. In such instances direct surgical approach should be used.

The present low operative mortality in cases of tracheoesophageal fistula, even when anastomosis of the two segments of an atresia of the esophagus is carried out, is due in part to improvements in surgical technique. A good deal of credit, however, must be given to a better understanding of the preoperative and postoperative care of the infant. He should be observed in the hospital for at least 24 hours. During this period of time it is important to evaluate his state of nutrition and hydration. If parenteral therapy is indicated, it should be used with understanding. Such infants should not receive unnecessary parenteral fluids. The over-zealous use of saline solution with resultant retention of the sodium ion is a common cause of postoperative edema in these small patients. Five per cent glucose in water, or, if the serum protein is found to be low, concentrated albumin solutions are of value. Complete blood studies are performed preoperatively. In general, if the red blood count is below 4,500,000, a small blood transfusion may be of assistance. If the red blood count is over this more or less arbitrary level, a transfusion of plasma alone might be considered. Each case, however, has to be decided upon its individual findings. Chemotherapy or antibiotic therapy is instituted prophylactically because of the always present or potential danger of pulmonary infection. Sodium sulfadiazine, penicillin, or aureomycin are given in adequate dosage, depending upon the size of the infant. Atropine is the only other preoperative medication. In cases where cyclopropane anesthesia is used, narcotics are found to be too depressing to the respiratory system of a young infant. When an adequately trained anesthetist is available, cyclopropane has been found to be the most satisfactory anesthetic agent. With this technique a high concentration of oxygen may be given constantly, and with an intratracheal tube in place positive pressure inflation may easily be resorted to if necessary. The advantages of positive pressure are obvious. Cyclopropane may be given either with a tightly fitting face mask or by an intratracheal tube. An intravenous cannula already inserted into an ankle vein before the operation begins is of great value before the operation is completed.

As a result of the pioneer work of Ladd,[5] Lanman[1] and Leven,[16] and the further clinical experience and research of Gross,[39] Haight,[4] Swenson,[41] Ladd,[40] Daniel[42] Humphreys[43] and others, vast improvements in this specialized thoracic surgical technique have been made, and the procedures are now more simplified. Their techniques have been carefully and adequately presented in the literature. Two surgical approaches have been used in performing the thoracotomy; namely, a retropleural or a transpleural exposure. The former method has the advantage of maintaining a sterile, intact pleural cavity so that infection or possible leakage from the site of the fistula does not cause significant trouble, as drainage is to the outside.

In the retropleural approach a curved incision is made on the right between the spinous processes and the scapula. The incision is curved downward and outward. Portions of the third, fourth and fifth ribs are resected posteriorly, but this causes no deformity as the bony defects in an infant are very quickly bridged by callous formation. The mediastinum is opened by reflecting the pleura from the thoracic cage and the azygos vein is brought into view as it traverses the esophagus. The vein is doubly ligated with black silk and divided between the ligatures. Some difficulty may be encountered in separating the trachea from the esophagus as these two structures are in intimate contact, especially in cases where a fistula is present. Dissection is then carried upward with care, and the vagus nerve must be constantly protected. When the fistula has been identified, it is freed up as completely as possible and doubly ligated with fine black silk. It is then divided between the ligatures and the severed ends of the fistula closed with a row or two of black silk sutures. A simple ligature may result in sloughing and the re-establishment of the patency of the fistula. For this reason a satisfactory closure is recommended in addition to ligation and division. Haight has suggested the repositioning of a bit of fat between the closed surfaces in order to prevent the recanalization of the fistula. He also advises the use of fine suture material and an ample cuff of tissue. A double row of sutures on the cuff of both the trachea and the esophagus is of value if sufficient room is available. Routine closure of the chest wound is employed.

In the transpleural approach a curved incision is made above the right breast and the pectoral muscles transected. The chest is entered in the third interspace by dividing the second and third costal cartilages. The pleura is then opened behind the phrenic nerve and dissection carried backward behind the esophagus. The trachea and esophagus are then carefully separated and the fistula doubly ligated, divided and closed in a manner similar to that used in the retropleural approach. The pleural flap is then sutured with interrupted silk sutures and the chest closed in routine fashion. Just before the skin is completely approximated, all blood and air are aspirated from the chest through a catheter which is quickly withdrawn as the last suture is tied. This helps greatly in the rapid expansion of the lung.

Postoperative care of these little patients is most important. They should be watched very carefully by special nurses adequately trained in the postoperative care of thoracic surgical patients. Adequate hydration is maintained but the circulation must not be overloaded with fluid. Again the warning regarding over-zealous use of saline solution must be repeated. Frequent determinations of serum protein should be made, and serum albumin or plasma therapy instituted if adequate levels are not maintained. An infant after closure of a tracheoesophageal fistula should be able to take fluids satisfactorily by mouth after recovery from the anesthesia. Chemotherapy and antibiotic therapy are continued to bring the patient through the immediate postoperative period, if possible, without the development of infection.

CASE REPORTS.

Case 1. L. R., Record No 315641 (courtesy Dr. Swenson) Birthday· March 7, 1947 This three-day-old baby girl was referred to The Children's Hospital on March 10, 1947, with the story that she had appeared perfectly normal at birth except for rapid respirations On the following day during her first feeding she experienced severe gagging, choking and marked cyanosis Further feedings precipitated the same difficulties, so oral feedings were stopped, and she was placed on parenteral fluids and intramuscular penicillin therapy She was then referred to The Children's Hospital for diagnosis and further treatment. At the time of admission the baby was lethargic, but well developed and well nourished She had a feeble, high pitched cry Respirations were rapid and mostly of the abdominal type. The breath sounds were diminished, especially at the left base, and occasional rales were heard throughout both lung fields The heart sounds were regular and the rhythm was normal There were no murmurs, and the heart did not appear grossly enlarged The reflexes were weak, and the baby seemed to be generally hypotonic

Shortly after admission lipiodol studies showed no obstruction of the esophagus, but halfway between the carina and the inlet of the thorax a definite fistula was visualized running obliquely downward from the trachea into the esophagus (see Figs 4 and 5). It appeared to be about 1 to 2 mm in diameter At the bifurcation of what appeared otherwise to be a normal tracheobronchial tree, the right upper lobe bronchus originated from the trachea (see Fig 6). Fluoroscopic examination of the chest showed a heart which was considerably enlarged and produced a slight posterior displacement of the esophagus The lungs were irregularly hyperventilated with moderate engorgement of vessels, but no definite aspiration pneumonia could be detected. It was felt that the baby had a congenital malformation of the heart with a probable shunt to the pulmonary circulation, possibly on the basis of a patent ductus arteriosus or an interauricular septal defect On the following day under cyclopropane anesthesia, thoracotomy was performed using the retropleural approach The esophagus appeared grossly normal The opening of the fistula into the trachea seemed to be about 3 to 5 mm., but was smaller on the esophageal side. It was ligated, divided, and the tracheal and esophageal openings closed. The baby seemed to withstand the procedure satisfactorily On the following day, however, she again developed cyanosis with feedings, and it was felt advisable to perform a temporary gastrostomy The attacks of cyanosis continued, however, in spite of only gastrostomy feedings Serum protein was only 4 6 mgm. per cent, so serum albumin was given in the hope of reducing the generalized edema which had developed She was kept on penicillin and streptomycin therapy and in oxygen most of the time The pneumonia became very widespread, and seemed especially resistant to treatment. The baby's course was downhill She died on the fifth day after closure of the tracheoesophageal fistula

Fig 4 Lateral Roentgenogram of intratracheal injection of lipiodol demonstrates tracheoesophageal fistula (A)

Postmortem examination showed a very extensive bilateral interstitial type of bronchopneumonia, as well as evidence of aspiration pneumonia (see Figs 7 and 8) There was also marked pulmonary edema The right upper lobe bronchus was found to originate from the trachea as had been noted in the Roentgenological findings The heart was grossly enlarged due to hypertrophy of its muscle, and there was a patent ductus arteriosus An incidental finding was a Meckel's diverticulum. The esophagus was normal, and the fistula had apparently been satisfactorily closed, with no evidence of mediastinal infection (see Fig 9—shows the histological aspect of the repair) The immediate cause of death in this case was felt to be the widespread bronchopneumonia which involved all lobes of both lungs (see Figs 10 and 11—demonstrate the organs of the neck and chest removed at the time of the postmortem examination)

Fig. 5 Close up view of Fig 4

Case 2 E. I, Record No 323888 (courtesy Dr Swenson) Birthday Sept 17, 1947 This five-day-old baby boy was referred to The Children's Hospital on Sept. 22, 1947, because of attacks of vomiting and cyanosis since birth Several hours after birth it was noted that he had an excessive amount of frothy saliva He was started on a formula which he promptly vomited and during the procedure became very cyanotic Vomiting and cyanosis were associated with further feedings, so hydration was maintained by parenteral fluids only He was referred to the hospital for diagnosis and treatment of *probable esophageal obstruction. At the time* of admission the baby appeared well developed and well nourished and in a fairly good state of *hydration He manifested a slightly wheezing type* of respiration, but the rate was normal, and there was no evidence of retraction of the chest or cyanosis. He had an increased amount of mucus in his mouth and pharynx, and numerous coarse rhonchi in both lungs.

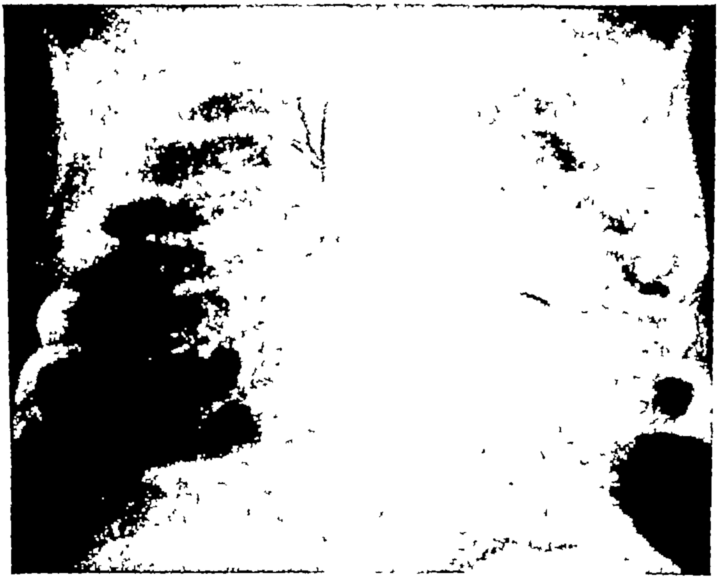

Fig 6 A-P Roentgenogram of intratracheal injection of lipiodol showing right upper lobe bronchus arising from trachea

Fig. 7. Photomicrograph (×160) of lung showing extensive bronchopneumonia with numerous macrophages in the alveolar spaces.

Shortly after admission the patient was given a swallow of lipiodol which showed an esophagus considerably narrowed at the fourth thoracic vertebra, but the lipiodol passed through this area into the stomach. There was a large amount of gas distributed throughout the intestinal tract. The lungs showed considerable peribronchial pneumonic infiltration throughout the entire chest, more marked in the right upper lobe, and probably secondary to aspiration. Some lipiodol was noted in the trachea and bronchi, but a fistula could not be demonstrated. The baby was kept on parenteral feedings and penicillin therapy. Because of excessive mucus which required frequent aspiration, coupled with several choking and gagging spells and a troublesome cough, it was felt advisable to perform a gastrostomy. This was done on the day following admission. An interesting finding is the fact that the stomach seemed to be so distended from time to time that it was necessary to deflate it by periodically opening the gastrostomy tube. Roentgenologic evidence of pneumonitis persisted although the baby's condition remained satisfactory. On the thirteenth day after the gastrostomy the esophagus was dilated by retrograde Tucker bougies, but it was possible to go through only a No. 10 Fr. Penicillin and streptomycin were given regularly in full dosage and oxygen and humidification were occasionally administered. Eleven days later his general condition as well as the Roentgenologic picture had improved, and it was again possible to dilate the esophagus, this time going through a No. 22 Fr. bougie. After the dilatation, further attempts were made to feed the patient liquids by mouth, but he still continued to have attacks of choking and cyanosis. It was generally supposed that he had a tracheoesophageal fistula which had not been demonstrated. The trachea and bronchi were then outlined by an intra-

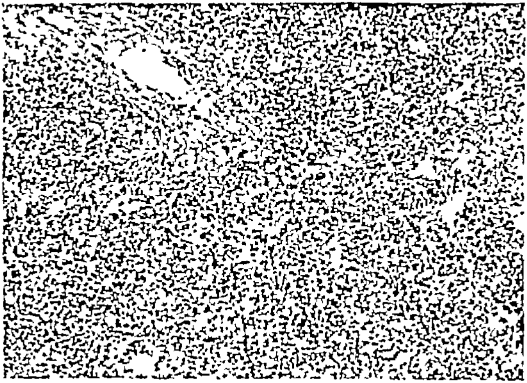

Fig. 8. Photomicrograph of lung (×120) showing bronchopneumonia, with marked edema and alveolar exudate

tracheal injection of lipiodol. No fistulous tract was demonstrated, but it was noted that "the lipiodol seemed to spill over into the esophagus." Two days later on a third attempt a narrow tracheoesophageal fistula was successfully demonstrated (see Figs. 12 and 13). It was 5 mm. in length, possibly 1 or 2 mm. in diameter, and ran from the posterior aspect of the trachea downward and backward, where it joined the esophagus at

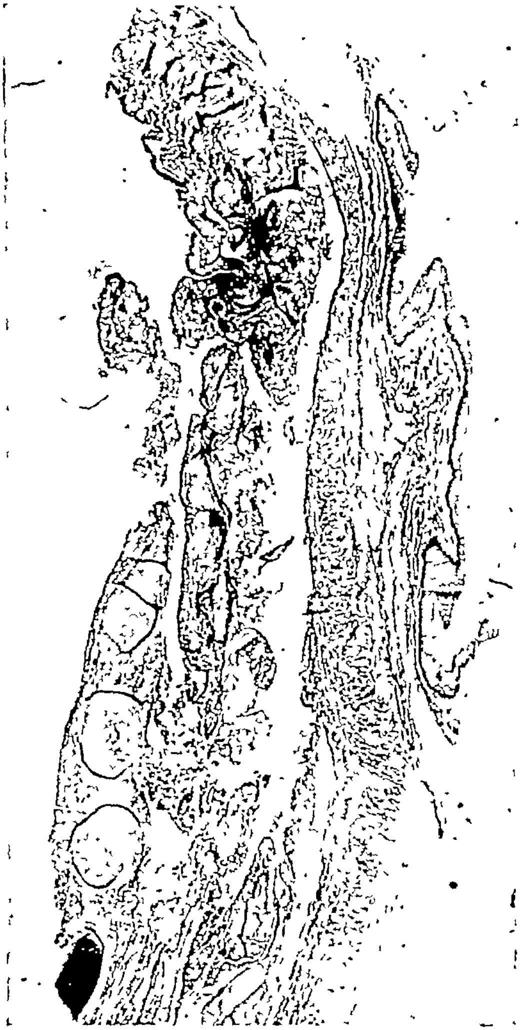

Fig. 9. Photomicrograph (×40) showing satisfactory healing at site of fistula repair.

the level of the second thoracic vertebra. Two days later under intratracheal cyclopropane anesthesia a thoracotomy was performed using a retropleural approach. The esophagus appeared grossly normal except for slight diminution in diameter in the region of the bifurcation of the trachea. It was tightly adherent to the trachea at this point, but it was possible to separate it satisfactorily The fistulous tract was demonstrated as outlined by the lipiodol, and was ligated, divided and the tracheal and esophageal openings closed with two rows of black silk sutures

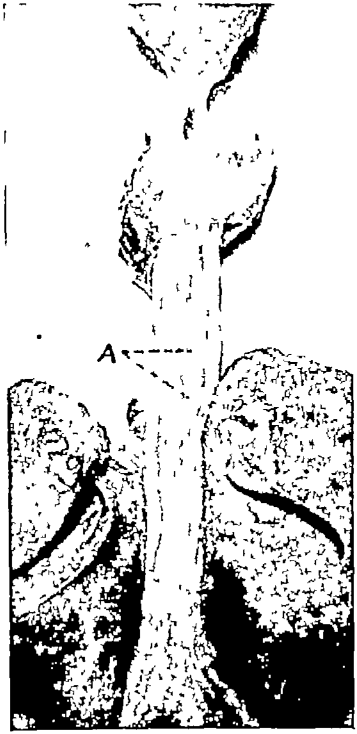

Fig. 10 Posterior view of autopsy specimen showing site of tracheoesophageal fistula repair (A) from esophageal side. Note edema of lungs.

The patient withstood the procedure satisfactorily and was discharged on the eighth day after the operation. Following the closure of the fistula he had no further trouble with excessive saliva and no regurgitation of feedings. The gastrostomy was closed three months later. He continued to gain weight and swallow normally. When he was last seen on July 11, 1950, at the age of two years and 10 months, there were no complaints, and he was able to take a perfectly normal solid diet without difficulty.

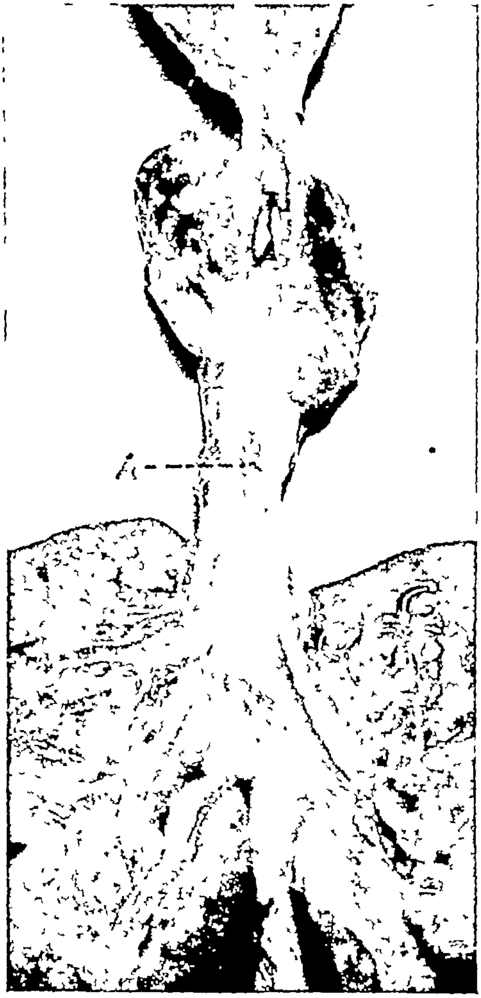

Fig. 11. Anterior view of fistula repair (A) from tracheal side.

Case 3 O. C, Record No 327688 (courtesy Dr Gross) Birthday Dec 16 1947 This one-month-old baby girl was referred to The Children s Hospital on Jan 17, 1948, for difficulty in swallowing and attacks of cyanosis She had been apparently normal until the third day of life at which time she choked during her first feeding Attacks of choking and cyanosis occurred with subsequent feeding On the fourteenth day of life a lipiodol swallow showed a filling defect in the posterior portion of the esophagus consistent with an aberrant subclavian artery She was referred to The Children s Hospital and made the rather long trip by plane

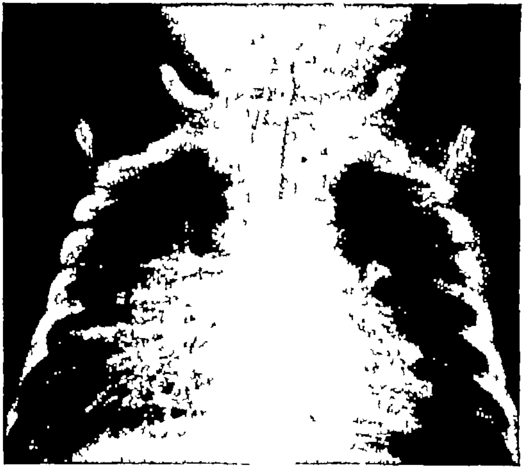

Fig 12 A P Roentgenogram of intratracheal injection of lipiodol out lining esophagus as well

During the flight she had one episode of cyanosis for which oxygen and metrazol were used The only other medication had been penicillin for the three days following the severest choking episode at two weeks of age At the time of admission the baby was well developed well nourished and apparently satisfactorily hydrated. Her respirations were somewhat rapid but the respiratory excursions were normal and the lungs were clear on auscultation and percussion The heart appeared normal on clinical examination.

Shortly after admission a lipiodol swallow revealed a posterior esophageal defect running obliquely upward from left to right at the level of the third thoracic vertebra. This was interpreted as the characteristic appearance of an aberrant right subclavian artery There was only slight delay in passage of lipiodol through the esophagus proximal to the defect and no evidence of obstruction. During forced expiration there was a flow of lipiodol into the trachea at the second thoracic vertebra through a minute fistula measuring at most 1 to 1.5 mm. in diameter (see Fig. 14). The trachea itself presented no displacement or compression. The lung picture was characteristic of aspiration pneumonia of moderate severity.

Because of the pneumonitis, after the patient had been on sulfadiazine and penicillin for two days, a gastrostomy was performed under cyclopropane anesthesia. The pneumonitis improved remarkably after this procedure. Forty-eight hours later, thoracotomy was performed under cyclopropane anesthesia given by an intratracheal tube. A transthoracic approach was used. The aberrant vessel was readily identified, doubly ligated and divided. The trachea and esophagus were carefully dissected and the fistula also readily identified and ligated. It was then divided, and the esophageal and tracheal openings closed. Blood and air were aspirated from the thoracic cavity through a catheter which was withdrawn just before the skin was tightly closed.

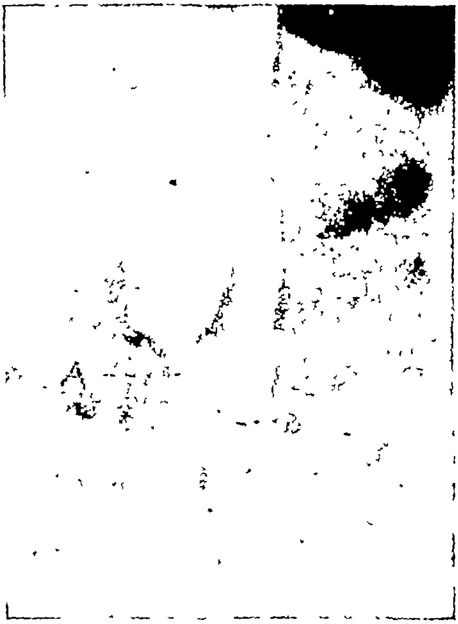

Fig. 13. Lateral Roentgenogram of same case. Note marked stenosis of esophagus (A) and fistula (B).

The infant had a satisfactory postoperative course except for a brief flare-up of the pneumonia in spite of continuing chemotherapy. Nutrition and adequate serum protein levels were maintained on gastrostomy feedings coupled with several blood transfusions. On the thirteenth postoperative day repeat lipiodol studies of the esophagus showed only slight persistence of the indentation in the posterior wall at the level of the divided subclavian artery. The notch was smaller and more shallow than on previous examination. The fistulous tract could not be identified. At the time of discharge from the hospital on the thirteenth postoperative day the baby was able to take many of her feedings by mouth. On the twenty-eighth postoperative day it was possible to remove the gastrostomy tube since the baby was swallowing perfectly normally. A two-year follow-up report was obtained in March, 1950. Her condition at that time was excellent, and she was taking a normal diet without difficulties.

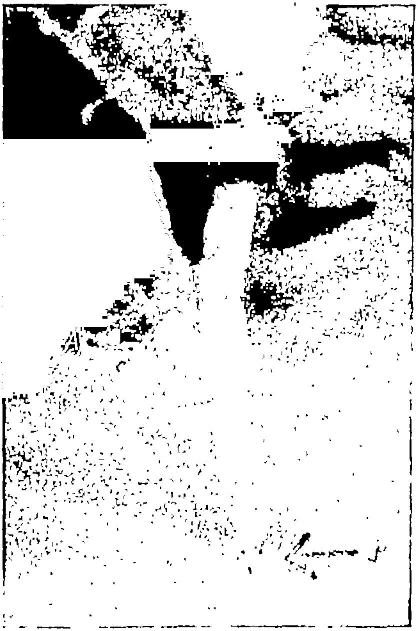

Fig. 14. Lipiodol Roentgenogram showing tracheoesophageal fistula (A) and posterior filling defect of esophagus produced by aberrant right subclavian artery (B).

Case 4: L. S. D., Record No. 224653. Birthday: Feb. 18, 1949. This 10-month-old baby boy was first seen in the out-patient department of The Children's Hospital on Dec. 17, 1949, with the story that since birth he had breathed heavily and noisily and seemed to have a constant upper respiratory infection. During the first month of life he had been kept in oxygen a good deal of the time. At three months of age he developed pneumonia and spent another month in a hospital. He had had no swallowing difficulties at any time. Whenever he started to gain weight, however, he developed another respiratory infection which resulted in further weight loss. He was slow in development and at the age of 10 months had no teeth and was unable to sit up alone. At the time of admission to the medical out-patient department of The Children's Hospital the baby was 10 months of age, but weighed only 12 pounds, six ounces. Height was 64.6 cm., and both weight and height were below the lowest percentile rating. His eyes had a definite Mongoloid appearance, and the fontanels were still open. The tongue was very large. The heart appeared somewhat enlarged, and the apex impulse was 1 to 2 cm. to the left of the midclavian line. The rate was 140 and there was marked sinus arrhythmia. There was a Grade II pulmonary systolic murmur, but no diastolic murmur was heard. The extremities showed marked general hypotonicity, the hands were wide and thick, and large spaces were noted between the first and second toes on both feet. The reflexes were equal and normally active. It was felt that the baby was Mongoloid and that he also had congenital heart disease. Fluoroscopic examination of the chest showed a right middle lobe pneumonia. There was right ventricular hypertrophy and the auricles were enlarged. The heart seemed to be increased in its transverse diameter and showed an unusual configuration with marked elevation of the apex. It was felt that the patient had an aberrant right subclavian artery and a left to right shunt through a patent interauricular septum. The patient was followed by his local doctor until the pneumonia had subsided and then was admitted to the hospital on Feb. 1, 1950, for more complete study. Because of the cardiac and vascular situation the baby was given a lipiodol swallow in order to determine whether there was any compression of the esophagus. A typical oblique filling defect was noted on the posterior aspect of the esophagus running upward from left to right at the level of the aortic arch (see Fig. 15). In addition lipiodol was seen to pass from the esophagus into the trachea through a definite fistulous communication on the anterior aspect of the esophagus just below the jugular notch. There was no esophageal obstruction, and in addition to vascular engorgement considerable irregularity of aeration of the lungs with atelectasis of the right upper lobe was noted. The heart was grossly enlarged both to the right and left and was abnormal in contour. There was a right ventricular hypertrophy with enlargement of both auricles and considerable fullness in the pulmonary artery area with engorgement of the intrapulmonary vessels. The appearance was that of an aberrant right subclavian artery arising as the last branch of the aortic arch and passing to the right behind the esophagus. It was felt that the patient in addition had a large interauricular septal defect.

Because of the findings of such a severe cardiac abnormality and the fact that the patient was a Mongolian idiot as well, it was decided that he was a poor surgical risk for operation upon either the constricting artery or the tracheoesophageal fistula. For this reason he was discharged to the care of his local doctor. On attempted follow-up examination a few weeks later it was discovered that the baby had died at home. No autopsy had been performed.

This baby represented a combination of several congenital anomalies. He had a severe malformation of the heart with an anomalous vascular system producing a probable right subclavian artery which because of its position caused compression of the esophagus. The baby from all physical appearances was a Mongolian idiot and in addition had a congenital tracheoesophageal fistula without an associated atresia of the esophagus Unfortunately, however, it was not possible to obtain positive confirmation of this latter finding either by surgery or by postmortem examination.

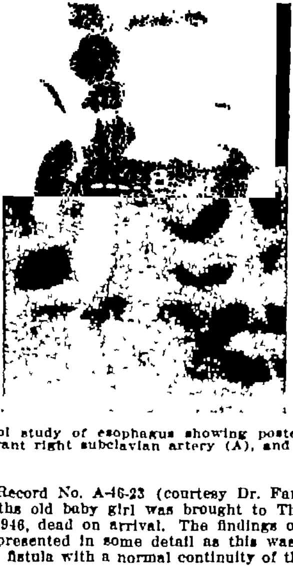

Fig 15 Lipiodol study of esophagus showing posterior filling defect produced by aberrant right subclavian artery (A), and tracheoesophageal *fistula (B).*

Case 5: C. F., Record No. A-46-23 (courtesy Dr. Farber). This seven and one-half months old baby girl was brought to The Children's Hospital on Feb. 6, 1946, dead on arrival. The findings of the postmortem examination are presented in some detail as this was the first case of tracheoesophageal fistula with a normal continuity of the esophagus seen at this hospital. The baby had been admitted to another hospital two days before death with a complaint of labored breathing. Six weeks previously she had been placed in a foster home and physical examination at that time was said to have been normal. Following placement the patient had been unable to swallow anything but liquids. There had been associated respiratory difficulty and a spasmodic nonproductive cough. Unfortunately further details of the history were not available. On the day of admission to the other hospital the baby was said to have been dehydrated and in acute respiratory distress. Roentgenograms showed a metallic foreign body interpreted as probably in the left main bronchus or possibly in the esophagus. Endoscopy was performed and during the procedure a foreign body was said to have been "dislodged but not removed." Transfer to The Children's Hospital was advised, but the patient died in the ambulance en route to the latter institution. The postmortem examination findings are as follows:

The body was that of a well developed and fairly well nourished female baby 61 cm. long (normal for this age is 65 cm.). No mediastinal emphysema or edema was noted. The lungs showed very extensive bronchopneumonia with little remaining aerated parenchyma. B. Friedlander, Type B, was recovered in pure culture, and the presence of gas bubbles in the blood stream was interpreted as being related to a probable B. Friedlander's bacteremia. Cultures from the blood stream, however, did not grow out any organisms. In the opinion of the pathologist there was no reason to believe that an air embolus had occurred before death. There was considerable patchy congestion of the mucosal surfaces of the lungs and large amounts of purulent material in the bronchi and bronchioles. On microscopic examination there was very little evidence of general aspiration pneumonitis, although in local areas the picture was rather typical. The diagnosis was that of a marked chronic purulent bronchitis. Acute inflammation and edema of the tracheobronchial lymph nodes were noted. Except for the local examination of the trachea and the esophagus, the only other congenital anomalies were double ureters and pelves of the kidneys on both sides, and a patent foramen ovale.

Six and five-tenth cm. below the proximal extremity and 6.8 cm. above the distal extremity of the esophagus was a fistula 0.4 cm. in diameter. The fistula was round and its periphery was smooth and regular (see Fig. 16A) .There was no gross evidence of inflammatory changes. It opened directly into the trachea just above the carina. In the region 0.7 cm. to the right and inferior to the fistula was an irregular, round defect in the esophageal wall 0.4 cm. in diameter. Just below the fistula was noted a small redundant flap of mucosa (see Fig. 17A) about 0.5 cm. in height and 0.5 cm. in diameter. The mucosa immediately adjacent to it was torn, eroded and necrotic. Lower down in the esophagus were several smaller longitudinal erosions (see Fig. 17B). On splitting the trachea anteriorly, the fistula was seen 1.3 cm. above the carina, measuring 0.4 cm. in diameter. At the carina and along the right borders of the trachea and esophagus were noted chains of lymph nodes which were dark brownish-red in color, moderately firm, and varied from 0.4 to 1.2 cm.

On microscopic examination of the irregular, ragged defect in the esophageal wall there was evidence of edema, acute and chronic inflammation, and necrosis. Numerous dilated.capillaries were noted growing toward the necrotic layer and these vessels showed hyperplastic endothelial lining. There was a rather long segment of mucosa including part of the muscularis mucosae torn away from the underlying layers and forming an elongated tab in the region adjacent to the erosion. The exposed surfaces were lined by a layer of fibrinoid necrosis in which were numerous colonies of bacilli.

Examination of the margin of the fistula into the trachea showed that the connective tissue layers of both the trachea and esophagus were continuous. The stratified squamous epithelium of the esophagus extended for some distance into the trachea where it merged into a thick, ciliated, pseudostratified columnar type of epithelium. There was slight edema associated with infiltration by lymphocytes and plasma cells, but no evidence of necrosis or an acute inflammatory cell response. This patient demonstrated a congenital tracheoesophageal fistula without atresia of the esophagus, and the perforation on the posterior wall of the esophagus below the fistula appeared to be traumatic and quite recent. It was considered that the perforation was the result of the fact that a foreign body had remained in the esophagus for some time and had eroded its wall, or that it was the result of the difficult endoscopic procedures at the other hospital. The foreign body must have been dislodged and passed through the intestinal tract prior to death At any rate, it was not discovered by careful search of the entire body at the time of the autopsy.

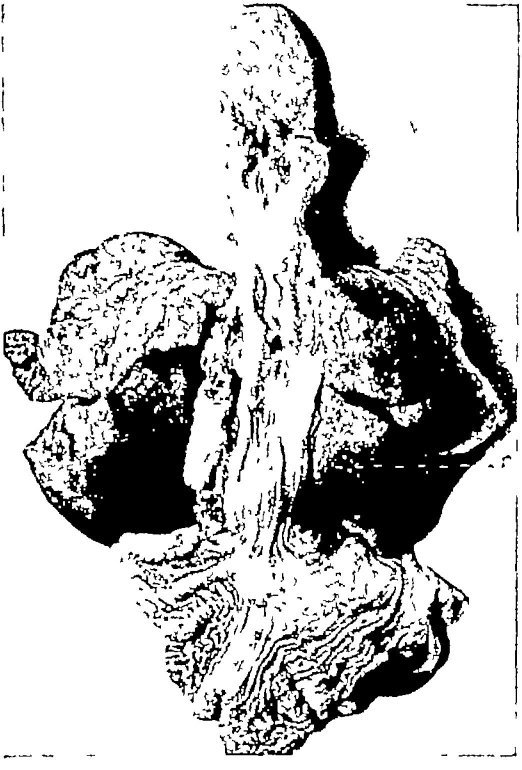

Fig. 16 Autopsy specimen of neck and thoracic organs opened posteriorly showing mucosal surface of esophagus Note fistulous opening (A) and yellow bronchopneumonic mottling (B) most striking on posterior surface of right lung

otherwise normal esophagus. Since 1946 four other cases reported in this series were diagnosed clinically, and three of them were treated surgically.

SUMMARY AND CONCLUSIONS.

1. Congenital tracheoesophageal fistula without the usually associated atresia of the esophagus is not so rare as the literature indicates. Only two cases have so far been reported in which the diagnosis was made and a successful operation performed. In neither of these was diagnosis made during infancy.

2. The embryology and possible causative factors are discussed.

    a. The most plausible theory is that there is an arrest in the development of the lateral cell masses which separate the primitive foregut tube into the esophagus dorsally and the trachea ventrally.

    b. A genetic cause of this arrest cannot be eliminated, and environmental factors should also be considered.

    c. The fistula is probably well established by the seventh to eighth week of fetal life (6 to 8 mm. stage) as embryological studies have demonstrated.

3. A brief description of the gross and microscopic pathology is included.

4. The symptomatology is not constant. Diagnosis should be suspected in an infant who has frequent attacks of choking, with or without cyanosis during feedings.

5. Diagnosis is often very difficult, and repetition of many of the diagnostic procedures is necessary. Methods employed consist of the passage of catheters, lipiodol studies of the trachea, and esophagus by film and fluoroscope, and repeated endoscopic procedures, possibly with dyes or with lipiodol under pressure. The passage of a catheter does not eliminate the possibility of a tracheoesophageal fistula.

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THE CLINICAL AND EXPERIMENTAL COMPARISON OF COCAINE AND PONTOCAINE AS TOPICAL ANESTHETICS IN OTOLARYNGOLOGICAL PRACTICE.[*]

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The purpose of this presentation is to discuss some clinical and experimental findings on the relative toxicity and potency of these two drugs which are so commonly used for topical anesthesia in the practice of otolaryngology. A survey was conducted in 1928 by the American Medical Association to compare the local anesthetics in general use at that time. Since then we have had the introduction of tetracaine, commonly known as pontocaine. A comparison of pontocaine with cocaine is all that will be discussed.

The problem was first approached as an experimental investigation by Dr. Eric Stark in the Dapartment of Pharmacology of the University of Toronto, under Prof. Ferguson. The clinical experiments were conducted in the Department of Otolaryngology, Toronto General Hospital. A survey was conducted by means of a questionnaire sent to the larger clinics in the United States and Canada.

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EXPERIMENTAL STUDIES.

The details of the experimental studies will not be given in this paper, but only a summary of the conclusions made from their analysis. The two drugs considered were used in their pure forms and in various strengths. They were also tested with their added preservatives as sold in the drug market. Local application to the larynx and carina by means of a specially designed laryngoscope and bronchoscope was used, and direct application by drop instillation after external isolation of the trachea. The experimental animals used were dogs, guinea pigs and rabbits, and many results were discarded because of factors which were considered to be due to extraneous causes. A summary only is given under the following headings: *1*. Measurements of toxicity. *2*. Fatal doses. *3*. Effects on mucous membrane. *4*. Estimation of the potency on human mucosa.

1. Measurements of Toxicity.

Reports on the toxicity of cocaine and pontocaine when injected into animals give figures for the median lethal dose of pontocaine which vary from three-fourths to one-third of those for cocaine.[1-3] In other words, the toxicity of pontocaine is given as one and three-tenths to three times that of cocaine. If these figures are applicable to humans, one would expect pontocaine to be a safer drug clinically than cocaine, since pontocaine is customarily applied in 1 to 2 per cent solution, or about one-fifth the concentration at which cocaine is ordinarily used.

We felt, however, that figures for lethal doses by injection were hardly fair for estimating relative toxicity of drugs used topically; consequently, an experiment was designed to measure the lethal doses of these drugs by application to the tracheal mucosa of the guinea pig.

Large guinea pigs, weighing 0.7 to 1.3 kgm., were secured in a supine position. The trachea was exposed through a small incision made after infiltration of the skin with a small amount of 0.5 per cent procaine hydrochloride. Cocaine as a 10 per cent solution of the hydrochloride, and pontocaine as 2 per cent solution of the hydrochloride, were injected into the trachea, drop by drop, through a 26 gauge needle on a tuberculin syringe. The rate of administration was timed to cause death with typical convulsions and respiratory failure in 10 to 15 minutes. The total volume injected seldom exceeded 0.6 cc., *i.e.*, not enough to drown the animals.

2. *Fatal Doses.*

The mean fatal dose for cocaine HCl (11 animals) was 24 mg./kg., with a standard deviation of 4.6 mg./kg. That of pontocaine HCl (11 animals) was 7.3 mg./kg. with a standard deviation of 0.2 mg. The toxicity of pontocaine HCl was thus found to be about three and four-tenths times that of cocaine HCl.

3. *Effects on Mucosa.*

In preliminary experiments with pontocaine injected into the trachea very slowly, the animals often died from respiratory obstruction. This proved to be due to swelling and desquamation of the tracheal mucosa. This unexpected result was obtained using many different solutions of pontocaine ranging in strength from 0.5 to 2 per cent, both freshly made and as supplied ready-made by the manufacturers. A similar desquamating action was observed on tracheal mucosa of rabbits. Although this peculiar irritating or histotoxic action has not been observed on human tracheal mucosa, it may be related to the clinical observation that pontocaine has a tendency to provoke asthmatic attacks in persons subject to this affliction.

In summary, these measurements of fatal doses support earlier ones in suggesting that pontocaine should have a greater margin of safety than cocaine when used at the customary concentrations. The reaction of the tracheal mucous membrane served a warning, however, that the acute lethal doses might not tell the whole story of potential toxicity.

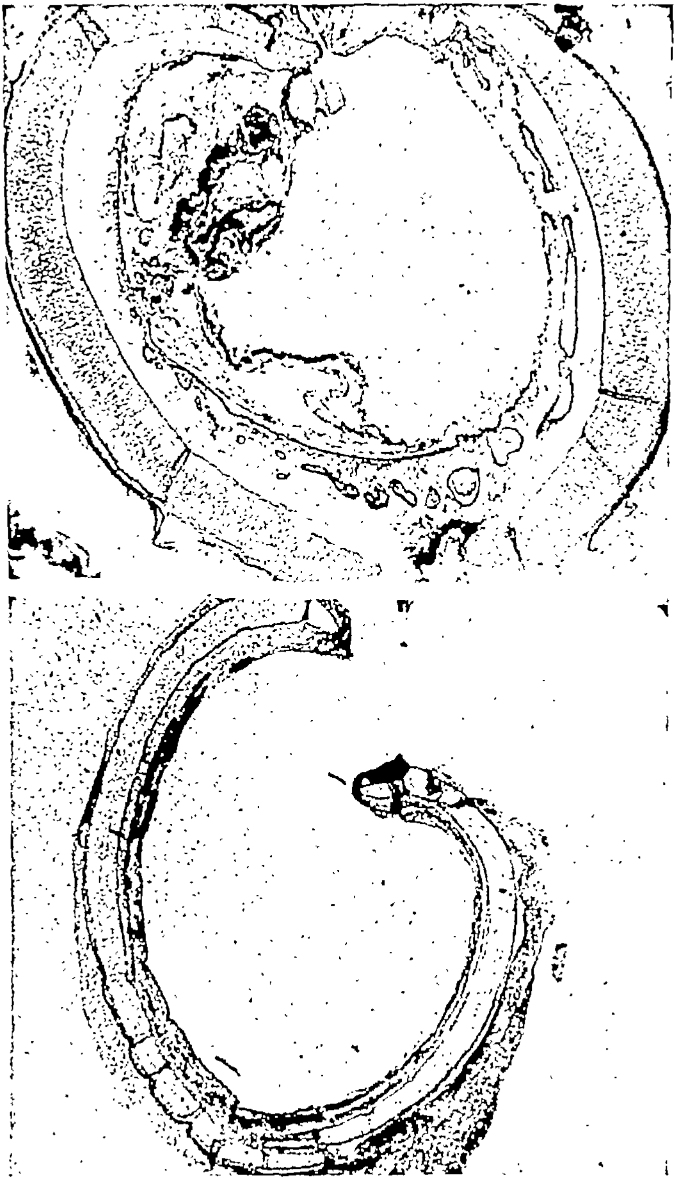

Fig. 1. Guinea pig trachea (low power). Section on left shows normal cross-section. Section on right shows edema and marked narrowing of lumen after pontocaine instillation.

4. Estimation of Potency on Human Mucosa.

Cocaine HCl 5 per cent was compared with pontocaine HCl 1 per cent by application to the nasal septa of patients. Pieces of blotting paper, half an inch square, were moistened with four drops of the solution under test and applied to healthy mucous membrane one-half to three-fourths of an inch behind

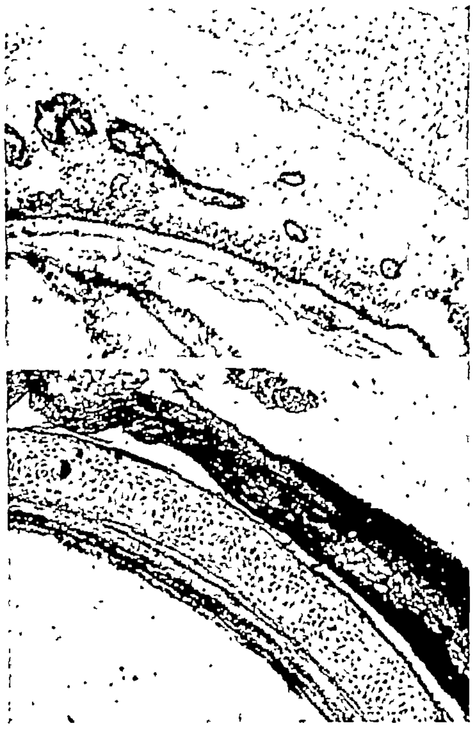

Fig 1 Guinea pig trachea (high power). Section on left shows narrow submucosa and normal ciliated epithelium. Section on right was after the instillation of pontocaine. It shows marked acute edema of the submucosa with very little inflammatory reaction except interstitial fibrin formation. The epithelium is in places denuded from the surface.

the columella. The area was tested with a sharp probe every 30 seconds till the pricking sensation disappeared. The paper was then removed and sensation tested every 30 seconds until it became normal again. Twenty patients were tested using pontocaine on one side of the septum and cocaine on the other, both at the same time.

In all but four patients the onset of anesthesia was more rapid with cocaine. The average time of onset was 3.9 minutes for pontocaine and 2.4 minutes for cocaine. The ranges were 1.5 to 4 minutes for cocaine and 2 to 7 minutes for pontocaine. The difference in speed of onset is significant ($p < .001$). The average duration of action was 7.6 minutes for pontocaine and 8.0 minutes for cocaine. The ranges were four to 12 minutes for each. Since the blotting papers were removed in each case as soon as the anesthesia was established, it follows that the time of application of cocaine was shorter than that of pontocaine. If the times of application had been equal it seems likely that the duration of action of cocaine would have been longer than that of pontocaine.

It may be concluded that 1 per cent pontocaine HCl has a potency either equal to or less than that of 5 per cent cocaine HCl, depending on what is considered a fair basis of comparison. In speed of onset the pontocaine was definitely inferior to cocaine solution in the strengths tested.

RESULTS OF QUESTIONNAIRE.

In choosing the clinics for our questionnaire it was felt that only the larger centers of the United States and Canada should be included. This was based on those clinics where a large volume of cases was to be found and where, also, the frequent use of these drugs would induce better technique in their application. Unfortunately some of the replies did not state, as requested, the number of cases treated and just said many thousands. These were discarded from the total of cases, but the complications were added so that these represent a group in a larger number of total cases and percentages cannot be estimated.

TOTAL NUMBER OF CASES TREATED, 39,278.

(With Cocaine and Pontocaine)

COMPARISON IN CASES WHICH USED COCAINE OR PONTOCAINE

Cocaine used exclusively	31,885 cases
Pontocaine used exclusively	7,394 cases
Total of cases reported	39,279 cases

CENTERS USING COCAINE IN TOPICAL ANESTHESIA

(Per Cent of Cases)

	%		%	
Boston	99.5	Philadelphia (1)	100	
U. Michigan	100	Philadelphia (2)	5	(allergic cases—bronchoscopy)
Vancouver	100			
U. Illinois	5	New Orleans	65	
Montreal	3	Toronto (1)	65	
Los Angeles	100	Toronto (2)	60	
		Chicago (2)	90	(uses both in each case with pontocaine spray first)

COMBINED USE OF ADRENALIN WITH PONTOCAINE OR COCAINE.

No conclusions or definite information could be recorded from the answer to this question. The amount used varied from equal parts to 1:4 in the nose and throat and a similar variance in the larynx and trachea. There is, however, a suggestion that the use of adrenalin might be an important factor in the faintness, rapid heart and vasomotor symptoms characteristic of the minor reactions. It is also a significant factor that several of the larger clinics, which use no vasoconstrictor drug, reported no reactions or deaths in their series of cases.

CONCENTRATION OF ANESTHESIA USED.

Cocaine.—Ten per cent in nose and pharynx, 5 per cent in larynx and trachea.

Pontocaine.—Two per cent in nose and pharynx, 1 per cent in larynx and trachea.

There were exceptions to this general rule. Most clinics insist that the quantity of pontocaine in trachea be limited to 2 cc. of 1 per cent solution. Various types of precautionary measures are used to assure the accurate measurement of this amount.

Untoward Reactions.—Under this heading it was our purpose to have a description of what was considered to be a reaction and we tried to classify these into three types: *a.* mild, *b.* severe (not fatal), *c.* deaths. The mild reactions were characterized by a fast thready pulse, fainting, sweating, pallor and other vasomotor symptoms. The severe reactions consisted of vascular collapse with changes in heart rhythm and convulsions. The deaths were due to cardiac failure or medullary failure.

CASES REPORTED WITH UNTOWARD REACTIONS

COCAINE		PONTOCAINE	
Mild	51 cases	Mild	36 cases
Severe	2 cases	Severe	7 cases
Deaths	3 cases	Deaths	4 cases

SUMMARY OF SURVEY.

Once more I must say that statistics are frequently misleading, but it is striking that in this large series of cases, from busy centers, that cocaine should be the anesthetic so generally used in preference to pontocaine. In view of this marked difference in the number of cases in which cocaine has been used it is quite significant that the deaths and severe reactions are more common with the use of pontocaine. There is little information gained from the rôle of adrenalin in combination with a topical anesthetic. A general agreement of the concentration of the anesthetic agent to be used in the various locations seems to exist.

Of timely interest is a survey made by Schindler[4] of 60 clinics doing gastroscopic examination in the United States, Great Britain and Australia. He reports on 22,351 gastroscopic examinations with only three deaths attributed to local anesthesia. All of these were from the use of pontocaine.

GENERAL SUMMARY.

Cocaine has been used and abused as a local anesthetic since 1884. It has been and is still used, in so many concentrations and by varying methods, that the accumulated knowledge of its good and also its dangerous effects has made it a relatively safe drug in experienced hands. We do know that experimentally it can be shown to produce topical anesthesia sooner and probably of longer duration than pontocaine. We have been shown by Leshure[5] and others that premedication with barbiturates reduces the toxic effects it can produce but does not preclude their appearance. We do know that individual hypersensitivity to the drug occurs so that small amounts, even as little as one-third gr. (*i.e.*, 20 mg.), according to Derbes,[6] may cause death. This hypersensitivity has been overemphasized according to our clinical survey. No satisfactory antidote is known to this type of reaction, although intravenous pentobarbital sodium relieves the convulsions and may control the toxic effect until the liver can perform its function of detoxication. According to Goodman and Gillman,[7] this occurs at the rate of one minimum lethal dose per hour.

It is not our purpose to discuss methods of safe use, but the suggestion of Whalen[8] that we have the patience to apply a small quantity locally by applicator or spray should help sort out, without a fatal termination, the very occasional hypersensitive individual. It has been pointed out by Titche that while scratch tests, patch tests or intracutaneous injections are satisfactory for drugs causing contact dermatitis, they are useless in drugs with other portals of entry. He concludes that when these deaths do occur with small doses, it is not due to an allergic response. Young[10] reports a remarkable case which they were able to follow by continuous electrocardiograph tracings during the progress of a fatal toxicosis. These showed auricular tachycardia followed by ventricular tachycardia and multiple ectopic ventricular beats before death.

There is some reason to believe that pontocaine, in the concentrations commonly used, is more irritating to the mucosal

4. A survey of some of the larger clinics in the United States and Canada has been made with reference to the use if these two drugs with the complications encountered.
5. Cocaine is by far the majority choice in clinical use.
6. Complications of a severe nature and deaths were found more frequently with the use of pontocaine
7. Cocaine has long years of useful service as a topical anesthesia and its vagaries are well known. It is still probably the most satisfactory of these two drugs to use in our specialty.

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PSYCHOSOMATIC MEDICINE AND OTOLARYNGOLOGY.*†

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> *I propose then that the physician endeavor to find out by all possible ingenuity of conversation what matter of anxiety there may have been upon the mind of the patient that has rendered his life burdensome. Having discovered the burden, use all possible means to take it off. Offer him such thoughts as may be the best anodynes for his distressed mind . . . especially the right thoughts of the righteous and the means of obtaining composure of mind. Give him a prospect if you can of some deliverance from his distresses or some abatement of them.*
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> *Essays to Do Good . . . Cotton Mather.*

It is probably an act of supererogation for a psychiatrist to appear before the Triological Society at this late date and to call attention to the fact that not only is our present-day concept of disease changing but that, also, disease itself is changing, for no specialty bears such eloquent testimony to this as does your own. Nevertheless, the things we have to say here need to be said at regular intervals, and if they prove unduly repetitious it cannot be helped, for, with Terence, we can only admit that "In fine, nothing is said now that has not been said before."

With the advent of the newer drugs and antibiotic agents, with the greater control of infectious diseases and epidemics and the greater increase in life span and life expectancy. the otolaryngologist, like other medical practitioners, finds himself confronted with an increased incidence of chronic disease

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†From the Department of Neurology and Psychiatry, Mayo Clinic.

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and emotional illness, manifested in protean forms and testimony to the status of our present-day culture. It is this latter group which claims our attention today—the emotionally distressed—as they present themselves in the offices of practitioners in the various fields represented here; but it would be well for you on another occasion to consider the emotional reactions of members of the older age groups, for before long a large segment of your practices will be concerned with this forgotten and neglected element in our medical population.

It is reasonable to suppose that the practitioners of your specialty see a large number of emotionally disturbed persons, for you work in an area important not only because of the special senses involved but also for cosmetic reasons. This is true because part of your field encompasses the facade of the individual which, attractive or not, is presented to his fellow man with varied emotions. My contention in this thesis is that you as specialists can do much to aid these distressed individuals, with their variety of psychosomatic complaints, and it has the added corollary that unless you do undertake the treatment of the milder conditions you encounter, many of them either will not be treated at all or, worse yet, will be treated by someone in a manner which, no matter how well intentioned, will serve only to fix the neuroses *in situ.*

There is no need for me to list the conditions in question; you know them well: the people whose noses are too short or too long, too thick or too thin, too wide or too narrow; those who are dizzy or who have "sinus," who have lumps in their throats or whose heads ache with varying intensity; the asthmatic, the "sneezers," those with coughs and allergies and all those who plague you by presenting symptoms enough but no organic basis to justify their complaints. You are better acquainted with them than am I, yet we should consider them together.

It has been said of medical specialists that their function is to lessen their importance as specialists and to strive to have their teachings incorporated into the body of medical practice. This effort is particularly necessary in the practice of psychiatry today, for there is general agreement among all concerned that even though psychiatrists are charged with the care of neurotic patients, most of the milder psychosomatic conditions will have to be treated by the practitioner or the specialist in whose field they fall, not only because they are in a much more favorable position to do so, but also because there will not be enough psychiatrists to assume this tremendous burden, even if that were desirable.

The thought might enter your minds that the already harassed otolaryngologist does not want this type of patient or practice, but the answer to this is—he already has them. Like God's poor, they are in plentiful supply, and after the gastroenterologist, it is probable that the otolaryngologist sees a larger number of them than does any other specialist. Some of these patients exhibit the frank neuroses and even the psychoses, while in others the psychiatric tones are subtly disguised. Many of them are "just nervous," but all of them are in need of help. Simply to designate their complaints as functional and to tell them to "go home; the troubles are all in your head" will not suffice—this is comparable to the psychiatrist telling them to go on a vacation, the trouble is all in their bodies. Either they need help from the practitioner they consult medically or they need to be put in the hands of someone who can understand and treat their ills.

Mrs. W., age 50 years, is an excellent example of the psychotic group mentioned above. For the last 10 years she has been making the rounds of otolaryngologists, dentists and oral surgeons. Her complaints are "pain in the mouth, tongue, cheeks"; "in the flesh" as she puts it. From physician to clinic, to dentist and surgeon she has made weary rounds. Most of them have assured her that it is "all due to her nerves" and have passed her on.

Very gentle and careful questioning reveals that under these complaints is an involved delusional system. She regards these pains as "just visitations" upon her. Actually, she believes they are all her husband's fault. Her delusional system includes people following her; people watching her; people asking questions which have multiple meanings; people talking about her; husband trying to kill her, and so on.

This patient has paranoid schizophrenia. It is useless to talk to her about her ideas being delusional. She counters by saying, "It can't be true; otherwise those other doctors would have told me so." The whole operation of diagnosing this illness takes but a few moments. Detected early, she would have been a candidate for insulin therapy; now it is probable that she will be a suicide, because she has given up hope and is depressed.

Mrs. X is not psychotic, but she does have a crippling neurosis. She is 36, an attractive person and her main complaints are "stiffness of the neck, tightness of the throat and difficulty in swallowing." She dates her illness to two years before her visit; it began the day her father died. She had been "his girl." Actually, she has been ill much longer than she suspected.

Careful questioning indicates that she has some insight into the causes of her illness. Her real trouble is a terrific antipathy to her mother. She is constantly fearful that she will betray her hostility; she is constantly holding herself in. No wonder she has stiffness of the neck and tightness of the throat.

As an only child, she was her father's pet. She was more at home with older people and could not compete with girls her age. She now looks down upon her friends and neighbors and feels superior to them. She remarks, "I am very ambitious." The relationship between her difficulties and her symptoms is obvious here, and even the patient recognizes it.

The emotional reactions of human beings are so involved in their symptomatology that the time has come when the physician can ill afford to know less about them than does the modern layman, and the layman's knowledge of the subject has greatly increased since the outbreak of World War II, for he can hardly go to the cinema or read a novel without encountering a psychiatric problem. A great deal has been learned in the last several decades from the scientific investigation of human nature, and some acquaintance with the more basic findings of psychosomatic medicine is indispensable for every practicing physician.

What is psychosomatic medicine? Even to mention the subject usually has been reason enough to start a fracas at almost any medical meeting. It is probable that one of the best definitions of what it is and what it implies is to be found in the journal, *Psychosomatic Medicine*,[1] in the introductory statement to its first issue. After noting that psychiatry's principal interest is the diseased mind, the statement declares that psychosomatic medicine covers a broader field and a different one. Its object is to study in their inter-relationships the psychologic and physiologic aspects of all normal and abnormal bodily functions and thus to integrate somatic therapy and psychotherapy. It designates a method of approach to the problems of etiology and therapy, rather than a delineation of the area. Briefly, it concerns itself with the psychologic approach to general medicine. "It is both a special field and an integral part of every medical specialty."

There are, of course, many misconceptions concerning psychosomatic medicine, and this is particularly true in its relationship to otolaryngology. To some psychiatrists, unfortunately, it has meant an undue emphasis upon *psyche* and not enough upon *soma.* To some otolaryngologists, the reverse has been true. Actually the concept means only to give each factor its rightful proportion and place. It is thus a point of view and a method of approach.

To utilize the psychosomatic approach, several things must be accepted as axiomatic. The first is that every disease has a psychic as well as a somatic component, and it is, therefore, necessary to know what sort of person it is who has the disease as well as what sort of disease the patient has. Second, we must regard disease as a process and not as a fixture or a series of end-results. Every disease process, whether it be designated "mental" or "physical," goes through a reversible or a physiologic change before it becomes structuralized and irreversible; therefore, the sharp distinction supposedly existing between functional and organic is extremely nebulous, for there is no one to say when functional disease ends and organic disease begins. A good example of this statement is cardiospasm. McMahon, Moersch and I[2] recently reported upon a group of 25 patients who had cardiospasm and who in addition to the necessary physical treatment underwent rather complete mental examinations. Twenty-four of them had suffered from emotional trauma at the time of the onset of their symptoms, and recurrences were noted after repeated or new psychic trauma. In general, the symptoms became aggravated when the patient was upset or "nervous." A number of environmental and emotional factors were noted to be present at the time the illness began. These included hostility and resentment, economic loss and insecurity, death of a parent on whom the patient was dependent, feelings of rejection, fears of death, insanity, failure and even surgical procedures.

As a group, these individuals gave evidence of personality difficulties in childhood plus neurotic difficulties in adult life. These findings bear out the contention that cardiospasm is a disease of the frustrated, and is not seen in happy, contented or well adjusted individuals. Yet, it is obvious that it would be difficult to tell when physiologic changes ended and structuralization began in their cases.

Third, we must include emotions in our list of potentially pathogenic agents, noting that they may become such under certain conditions and that when these conditions occur, conflict is set up. This conflict may be so painful that it is excluded from consciousness. This should not be new to us, for it was described by the physiologists, Pavlov and Cannon. John Hunter demonstrated it graphically when he made his famous declaration that his life was in the hands of the rascal who made him angry, and then proved it later by dying in a fit of rage.

Fourth, we must be willing to admit that a potentially injurious agent, be it emotional or bacterial, cannot bring about disease unless the organism is prepared for it in a manner physiologic, psychic, or both. Experiment has shown that emotional stress seriously limits the ability of the organism to regain a stable equilibrium after it has been subjected to some strain or injury.

If we are willing to accept these axioms, then the next step requires us to make an effort to understand the individual and the manner in which he reacts to stress. To paraphrase Dr. Alan Gregg—we must not consider the personality of an individual to be merely an inconvenient appendage to the particular organ we are studying. To understand people, we must be willing to sit patiently and to listen to them, and while we are listening to what they are saying, we must surmise what it is they are not saying and to judge how it is affecting them. We must expect our advice to be eagerly sought and then not followed; yet, in the face of this and numerous other frustrations we are required to exhibit that attitude of equanimity which Sir William Osler held to be one of our essential medical virtues.

This concept of the physician-patient relationship is no psychiatric mumbo-jumbo. It is one of the most essential ingredients of every therapeutic process. We cannot blithely dismiss it as mere bedside manner, for in every meeting of the physician and patient there is either a psychotherapeutic or a psychonoxious influence at work. From the initial interviews until the last meeting, in the physical examination and in the prescription of drugs, emotional and other cross-currents are playing a rôle. Upon a physician's ability to direct these cross-currents hinges a large part of his success.

At times, patients tend to be overdependent upon physicians and at other times to seem hostile or belligerent. The doctor's task is not to respond to either of these attitudes, for they are not meant for him personally. They are directed at what the physician stands for in the mind of the patient. Sometimes the patient tends to regard the doctor as a minor deity, but this is not harmful unless the physician develops the belief that this attitude is correct and justifiable. Then his usefulness to the patient is at an end. At other times, when the patient is hostile, the hostility is not a personal thing either, and to respond to it by becoming angry removes the possibility of helping him. The only time a physician becomes angry at a patient is when he, himself, is frustrated, and he should understand that patients' reactions to him usually are conditioned by their relationship to authoritarian figures early in their lives, especially parents and teachers.

In addition to what the patient thinks of his own illness, what he thinks of the physician who treats it also is important. In regard to the former, one thing is certain: the patient rarely is objective about his illness. He presents anxieties, fears and physical symptoms. He wants, or thinks he wants, treatment and whatever help is available. He may readily admit he is nervous or in fact that the whole family is nervous, but he fails to equate these facts with his presenting symptoms. While the whole situation may be obvious to the physician who can readily relate psychic and somatic elements, the patient does not see the connection or if he does see it, he sees it intellectually without feeling it. These problems which he has disguised as "symptoms" are sources of deep and painful anxiety to him.

Mr Y is an example of this type of reaction Twenty-eight years old and unmarried, he had been a regular visitor to the out patient department of a large city hospital His chief complaints were headache, which he insisted was due to "sinus," and some abdominal pain Although he was studied carefully, no organic disease was found. The symptoms did not respond to treatment, and had progressed to the point where the headache now interfered with his work. This man's illness had begun four years previously When he was first questioned regarding any possibility of trouble at the time of the onset of his symptoms, he stated that he had been much excited and upset about a personal situation, but he did not see why he should go into that, for it had nothing to do with his headache and abdominal pain Further questioning brought out the following facts:

Four years previously the patient had been haled into court along with several other young men in his neighborhood One of the young ladies in his locality had borne a child, and its paternity was to be decided in the municipal court. Over his violent protestations, he had been adjudged the winner of the contest, and along with the doubtful honor of fatherhood which was conferred upon him, he also was awarded the privilege of supporting the child until it was 16 years of age

In the ensuing period of emotional turmoil he commenced to have difficulty with his fellow employees and also to hate his job He was now in the anomalous position of making money in a job which he hated, and paying part of it to a girl he hated even more He was in a dilemma He could not run away, he was under court order and in addition, was the only support of a widowed mother

One other problem arose to plague him In an unguarded moment he had discussed matrimony with another young lady, and she was now busily engaged in making plans for the wedding His ideas on this subject had changed, but he said he did not want to hurt her, so he guessed he would have to go through with it.

Although the patient insisted that the problems which he related had no connection with his headache and abdominal pain the relationship is obvious Furthermore, he appeared to have enough difficulty to keep him supplied with headaches until the child reached the age of 16 It is also obvious that illness might offer a way out of his unpleasant situation

The solution, from a psychiatric standpoint, might come about as follows *1* If John is ill he cannot work, this takes him away from an unpleasant job *2* If John cannot work, he receives no pay and it is impossible for him to support a child, despite the court order *3* If John is sick and cannot work, it obviously is unfair for him to marry and expect his wife to work and support him Thus, the third unpleasant possibility facing him is removed

No amount of local treatment would have cured him of his headache The above mentioned method of approach to the problem admittedly superficial, is within the range of every practitioner

An important thing for us to keep in mind, in regard to the foregoing patients and in fact in all patients, is that their neuroses have meanings. They do not develop neuroses simply to annoy or harass physicians. They follow a purposive pattern in their symptoms, and this pattern represents the individual's reaction to the situation within himself or the surroundings which he finds difficult and unmanageable. His method of reacting to situations is his very own, and it is determined by his personality and by his previous experience. His reactions represent a compromise between the different arguments of his personality or between his personality and environmental influences. Ideas which become too painful are repressed, but the tensions they engender remain. When the stress of these varied emotions becomes too great, they appear in the form of symptoms, often in symbolic form, and hence the patient's neurotic manifestations frequently give a clue to the underlying difficulty.

It is evident, then, that a number of patients who come to the otolaryngologist for diagnosis and treatment present symptoms which are nothing more than the regular physiologic manifestations of anxiety. With the inevitable tendency toward self-diagnosis, these symptoms may be formulated by the patient into "sinus trouble" or "dizziness" and when, through introspection and suggestion, such a patient has added a few layers of his own ideas of what a sufferer with these diseases should have, he presents a complicated problem. During the examination one has an opportunity to observe the moist palms, the tremors, the wide pupils, the perspiration and the general restlessness which betrays the presence of anxiety. Miss Z typifies members of this group.

The patient is 39 years old; she was divorced after an unfortunate and short-lived marriage. Her complaints are of tinnitus and dizziness. Careful physical, neurologic and otologic examinations fail to reveal organic disease, but all consultants agree that she probably had Ménière's syndrome at the inception of the illness. The patient obviously is under stress and shows all the symptoms of anxiety mentioned above. Upon questioning, it develops that her husband was uncouth and brutal, and that the patient was pushed into the marriage by family pressures. The husband left her after infecting her with a venereal disease. Upon her return home the patient withdrew from her friends and society, fearful that people were critical of her. She had resisted attempts of her husband to initiate her into abnormal sexual practices. All this was the proximal background upon which the neurosis could develop. The remote background was an insecure childhood in a foreign settlement. The exciting cause of the emotional upset was the overhearing of a conversation through the consulting room door in a physician's office. From this conversation the patient got the idea that perhaps she was abnormal sexually. At approximately the same time, a man in the neighborhood was arrested for molesting children. The patient made an unfortunate connection in her mind, and compared this man with herself. All her old insecurities were mobilized, and the patient feared to leave the house lest she be pointed out as abnormal. Her first attacks of dizziness occurred at this time, and she began the rounds of physicians, seeking help. As her anxiety increased, her dizziness became worse and her illness was soon in a vicious cycle. After several interviews and an understanding of her fears and panic, her convalescence began.

The situation typified above and also admittedly oversimplified, gives an indication of the involved psychopathology which must underlie and accentuate the ordinary complaints which patients bring to us, and which exist in the absence of any organic pathologic process. The focus of these difficulties was determined by the original slight infectious process. To seek out the causes of psychosomatic problems, we need the answers to three questions:

1. What were the original characteristics of the personality of the patients in question?
2. Why did they become ill at the particular time they did?
3. Why did they become ill in the manner in which they did?

If we know the answers to these three questions, we shall have taken the first step toward understanding psychosomatic manifestations and, as someone has indicated, the only illnesses which are not psychosomatic are those which lie in the realm of veterinary medicine. It is seldom, however, that we obtain the answers to these questions in a short or superficial examination. The answers must be obtained by gentle, careful and thorough investigations into the psychologic background of the individual. Any attempt to badger the patient to produce satisfactory answers is foredoomed to failure.

As mentioned at the beginning of this discussion, all that one can hope to accomplish in a resumé of this type is to renew the interest of the otolaryngologist in the psychosomatic approach to problems, and to bring some of its elements into sharper focus. This leaves the whole question of treatment unanswered, but fortunately the various available psychotherapeutic methods are now outlined and simplified in numerous publications intended for the general practitioner. The main thing to keep in mind is avoidance of meddlesome psychotherapy and the stirring up of anxiety which is latent. No one ever got into trouble of this kind by listening patiently, questioning carefully and formulating the problem simply and with understanding in his own mind.

The question of symptomatic treatment was mentioned in the discussion of the illustrative cases. Lest there be some misunderstanding and these allusions be thought to be critical, it should be noted that we recognize symptomatic treatment not only to be justifiable, but in some situations undoubtedly to be the treatment of choice. Not only does it enable the physician to alleviate troublesome symptoms, but at times it acts against the basic difficulty itself. In some cases it breaks up the vicious cycle in which the pathologic process conditions certain alterations, which alterations in turn further the progress of the disease. Although this approach admittedly is not curative, it may restore a balance which influences favorably the patient's feeling, tone and capacity to work.

Not even in the use of the new hormones, ACTH and cortisone, in the treatment of the various otolaryngologic conditions for which they might be indicated, can you afford to forget the psychosomatic approach, for the administration of these hormones frequently has been associated with subjective and objective psychologic changes. Rome and I[3] have graded these changes as follows:

Grade 1 includes the improved emotional tone as expressed in the responses of those who express their relief in their speech and behavior. It seems in some to be the direct consequence of a rejuvenated hope.

Grade 2 includes the responses of patients who showed a quantitative elaboration of this reaction. They are greatly stimulated in their thinking and behavior, and in some instances are accelerated to the point of mental excitement, restlessness and a rapidly fluctuant mood. During periods of elation they tend to be facetious and even silly.

Grade 3 is comprised of the responses of patients whose personalities before the administration of cortisone or ACTH showed evidence of serious psychologic conflict which was poorly managed. Within a short time after the administration of the hormones, these conflicts were fulminated, and the physician then had a disturbed individual on his hands.

Grade 4 is a relatively unusual response. The clinical picture is an overtly psychotic one of unusually brief duration. Its character is determined by the patient's prepsychotic personality.

These psychologic changes, all of them relatively short-lived and all of them reversible, probably are due to the sudden and profound alterations, the *milieu intérieur,* caused by the hormones and the consequent effect upon the patient's metabolic homeostasis. The variety and degree of this reaction are due to many causes, and collectively they constitute a major stress, as taxing of his psychologic adaptation as any serious external stress would be to an equally vulnerable person; therefore, the possibilities of untoward reactions will have to be kept in mind when these hormones are administered.

There are many other facets to the psychosomatic problem, for we cannot practice medicine in a vacuum. Before long we shall have to begin to pay more attention to the *milieu* in which the *psyche* and *soma* operate, and this will necessitate sociologic considerations. This has nothing to do with social medicine; it means simply that the individual is strongly influenced by his environment and social surroundings.

This is never more patent than it is in military medical practice, in which some of the younger otolaryngologists will soon find themselves. I am convinced that the touchstone of military psychosomatic conditions is in motivation and morale. It makes little or no difference in many of these patients how severe is the neurosis from which they suffer if they are properly motivated. The cure of their psychosomatic symptoms, therefore, lies in prevention of these symptoms, and this prevention lies in proper motivation. This proper motivation should be developed in our high schools and our colleges, but to go into that would take us too far afield.

One cannot see the things we have spoken about here today through a head mirror. They are the larger aspects of the problem, and yet they vitally concern each one of us here, for they speak of factors which influence our patients. In the services they influence our fighting men, and the importance of keeping the latter group in good health becomes more apparent every day.

Now, somewhat fearful that I have meandered about and tread injudiciously upon medical toes, I desire to offer an olive branch. If I needed your ministrations, I would not want you to retalitate by offering to engage me in sociologic discourse. I close this dissertation in the words of Burton, who in his "Anatomy of Melancholy," also became perhaps justifiably timorous, being fearful of the future treatment he might get. He said: "I will urge these cavilling and contumelious arguments no further lest some physician should mistake me and deny me physic when I am sick."

For my part, I assure you I am well persuaded of physic and of the otolaryngologist's art

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SECRETORY EFFUSION OF THE TYMPANUM.*

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The title of this paper has been chosen because it suggests a sterile secretion in the middle ear or mastoid cells. Any one or both portions of this system are frequently involved.

Various terms have been used to describe this condition but perhaps the ones most commonly mentioned are hydrops of the middle ear and secretory or catarrhal otitis media. The two latter terms give the impression of an inflammatory process in the middle ear, and while a mild inflammation often initiates the condition the fluid behind the drum is almost invariably sterile.

Although a number of articles have appeared in recent years on "Secretory Otitis Media" the condition was recognized and accurately described by Politzer about 80 years ago. Quoting from one of Politzer's students — "In secretory catarrh the liquid is often brownish in color and this gives a yellowish tinge to the drum. The membrane frequently appears shiny as though the patient instilled a little oil in his ear. The drum is often infiltrated with fluid."[1]

About the same time that Politzer[2] described exudative catarrh of the middle ear, Sir William Wilde wrote his book on "Aural Surgery." An Irish pioneer in otology, Wilde made many observations but is perhaps best remembered for his classical "Wilde's incision" for acute mastoiditis. Although he carefully described the incision behind the ear, he never actually opened the mastoid cells. According to Stevenson and Guthrie,[3] "Wilde did not practice paracentesis for acute

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otitis, but in cases of so-called Eustachian obstruction he made an artificial perforation of the tympanic membrane, cauterizng the edges with nitrate of silver to ensure patency."

The apparent prevalence of this condition, as shown by the number of recent articles,[4-11] is in all probability due to the widespread use of chemotherapy. Since the advent of the sulfacompounds in the middle thirties and penicillin in the early forties, undoubtedly many cases of acute nasopharyngitis have been checked which otherwise would have developed suppurative otitis media. It has also been suggested that virus infections are often responsible. The increased popularity of plane travel has also shown a greater number of cases of aerotitis, sometimes with effusion. As a result patients frequently are considered cured, who still have a residual exudate in the middle ear which is free of organisms. The fluid may remain for days or months and varies in amount in different patients. Under the care of an able otologist this condition should not be allowed to persist.

Geographically, secretory catarrh does not appear to be limited to any particular area; it is widely distributed and found in all ages. Climatic conditions seem to have little bearing on its frequency, although during the Fall and again in the Spring, when changeable weather is the rule, acute upper respiratory infections are more prevalent, and effusion into the tympanum seen more often.

Etiology: An inflammation of the nasopharynx producing obstruction of the Eustachian tube is the most frequent cause of effusion into the middle ear. The swelling may be limited to the beginning of the tube or involve the whole tube and tympanum as mentioned above. The chief causes of this condition are so well known that repetition is unnecessary. They can all be summed up under the term obstructed noses. Whether the nasal obstruction is mechanical in origin or due to infection or allergy makes little difference. The result in the tympanum is identical. Two important causes in adults which should always be kept in mind are malocclusion of the teeth and nasopharyngeal growths.

The importance of tumors of the nasopharynx as a cause of effusion into the tympanum cannot be emphasized too strongly. During the preparation of this paper a case presented itself which gave a typical history.

A married woman of 46 years complained of a blocked sensation in the left ear. This symptom had been present for the previous six months and she had been treated elsewhere, the tube being inflated 18 or 20 times. The hearing returned to normal after each inflation, but this temporary improvement lasted only for a day or two. The left drum was markedly retracted and showed a dull reddish color—the right drum being normal. Inspection of the nasopharynx with the nasopharyngoscope and postnasal mirror showed a roughening of the tissue around the Eustachian tube as compared to the normal tubal opening on the right. A biopsy under local anesthesia proved to be squamous celled carcinoma. X-ray films of the base of the skull showed marked erosion of the basisphenoid. The patient was given a series of X-ray treatments but one cannot help but feel that valuable time was lost. In relating this case two facts should be stressed The catarrhal deafness which recurred within a day or so after inflation should have made the otologist suspicious. The very nature of the temporary improvement should at once have prompted a thorough examination of the nasopharynx. This neglected area should, of course, be investigated with all the means at our disposal—if necessary, after cocainization. As a general rule cervical adenitis is the earliest complaint in nasopharyngeal tumors,[12] but fullness in the ear with conductive deafness, otalgia and tinnitus which persist should also be kept in mind as possible symptoms.

Although effusion into the tympanum was known and described in great detail well over a half century ago, a few practical points might be mentioned that have not been noted in recent articles.

Pathology: According to Prof. Ruttin, of Vienna, who was a pupil of Adam Politzer, catarrhs of the middle ear are divided into *A*. acute catarrh, *i.e.*, *1*. dry, *2*. moist, and *3*. secretory catarrh; and *B*. chronic catarrh. "They are characterized anatomically by only slight hyperemia, swelling and tumefaction of the mucous membrane, and by the extravasation of a clear, serous, or viscid, sticky mucus exudate into the tympanum."

In dry catarrh, the drum membrane is retracted, but there is no secretion in the tympanum or even in the mucous membrane lining the cavity. The mucosa may appear quite normal.

In moist catarrh, there is secretion in the mucous membrane, but no fluid in the tympanum. The lining mucous membrane is swollen and edematous.

If the process goes on to the stage of secretory catarrh, free fluid appears in the tympanum. As mentioned previously, the fluid is either thin and serous in consistency or thick and viscid. The thin serous effusion, which is much more common, is thought to be due to negative pressure and is more in the nature of a sterile transudate.

The thick, sticky mucous secretion is more of an exudate and varies in consistency depending upon the amount of mucus present. Serous fluid has a high protein content which coagulates readily when exposed to the air, while mucus fluid has a low protein content. In both cases the fluid is sterile.

According to Ruttin, dry catarrh is found in cases of slight colds, especially when the anterior parts of the nose are affected. In such cases the drum membrane is grayish and retracted. Moist catarrh results from inflammation in the posterior parts of the nose and nasopharynx, such as occurs in children with enlarged tonsils and adenoids and in adults with posterior tip hypertrophies or swollen lateral bands. In this type, the drum markedly retracted often shows a reddish tinge, due to swollen mucous membrane shining through. The retraction is thought to be the result of a mild inflammation of the tensor tympani muscle which pulls the membrane inward.

Often in cases of secretory catarrh where the Eustachian tube is closed, the process is not limited to the tube but also involves the middle ear and all connecting mastoid cells.

Symptoms: The patient frequently complains of a blocked sensation in the affected ear with dullness in hearing and sometimes a lack of resonance to the voice. There is usually a history of a previous cold, and tinnitus is common.

Often the patient says his voice sounds as though he were "talking in a barrel." The degree of deafness and fullness depends upon the position and amount of secretion present. A patient often volunteers the statement that the hearing is dull while lying in bed and improves on assuming the upright position. In the recumbent position the oval and round windows are covered, impairing the hearing; but on arising the fluid sinks to the bottom of the tympanum, and the hearing improves. At times patients complain of crackles in the ear due to bubbles of air in the fluid.

Frequently both ears are affected. Although occasionally symptoms are indefinite or perhaps lacking altogether, every experienced otologist should be able to diagnose and treat these cases.

Signs: The appearance of the drum varies, depending upon its thickness or transparency, the amount and color of the exudate, duration of the course, and the degree of reddening of the tympanic lining membrane. When enough secretion is present to show a fluid line or meniscus across the drum the diagnosis is relatively simple.

The variations in color and position of the fluid level are well known and have been described frequently. An exudate which gives a yellowish or amber color to the drum below the fluid level is as a rule thin and serous, rather than heavy and viscid.

One or two other observations of Ruttin are worth mentioning: "When the fluid level line is below the umbo of the malleus it usually goes across the entire drum membrane. When it is above the end of the handle it generally goes half way. If in doubt about the fluid level always inflate the tubes or move the head from side to side to change the level line. When the hearing is not improved by air inflation, the case is not secretory catarrh."

There are exceptions, of course, when air inflations do not appear to improve the hearing and in these occasional cases one is justified in doing a paracentesis. It is much safer to incise the drum when in doubt than to leave secretion in a tympanum which may later result in bands and strands of fibrous tissue binding down the ossicles and producing adhesive deafness in later years.

This is particularly true in children. The cases most frequently undiagnosed are those in which the tympanum and perhaps the mastoid cells are filled with fluid. In the absence of a fluid line one has to depend upon the color of the drum to diagnose the presence of secretion. The range of colors has been variously described as yellow, amber, greenish-yellow or red, sometimes verging on a reddish purple. When the yellow or amber color is seen, it is more marked behind the umbo, *i.e.*, over the promontory. Often in these cases a fluid line will appear after air inflation. When the drum is one of the above shades the color is well marked with more luster than usual and the retracted handle of the malleus is narrower and very clearly defined. (In the normal malleus, two-thirds of the thickness of the handle is connective tissue, and the fluid behind the drum makes this tissue transparent. This accounts for the narrow appearance of the handle.)

Auscultation is often an aid in making a diagnosis. To those accustomed to using the Eustachian catheter the sound of air through the tube is very characteristic. Any abnormal sound, whether it is a crackling noise, bubbles, a series of squeaks or a faint thud, should make one suspicious of fluid in the middle ear. If the drum before inflation or catheterization appears thickened and retracted with a very prominent short process, the diagnosis is more definite.

A Siegle's speculum is also of value. When suction is applied in the external meatus by means of this instrument the hearing is often improved. Sometimes a fluid level appears which was not present before using suction. As mentioned, if there is any question of fluid in the tympanum after a detailed history and careful inspection an opening should be made in the drum. Whether this is done by means of a hypodermic needle or a paracentesis knife is a matter of choice.

A No. 22 or perhaps a larger No. 20 needle, two and one-half to three inches in length, with a short bevel of 1 to 1.5 mm., is usually sufficient. Many able otologists object to this method, as the opening often tends to close too quickly and has to be repeated. They prefer to incise the drum to allow even viscid fluid to exude more freely. There is a marked difference of opinion as to where the incision should be made. This will be dealt with under Treatment.

In most cases the drum can be incised without anesthesia, even in young children. The objection has been raised that a local anesthetic on the drum increases the chance of infecting the middle ear. This has not been borne out by experience. Unless the knife touches the promontory on the inner wall there is little discomfort. This has been mentioned previously many times.

A preliminary inflation of the tube raises the intratympanic pressure so that on opening the drum the contained fluid is often forced into the meatus.

Most otologists seem to prefer inflation and spot suction to the use of the Siegle's speculum. Applying suction directly to the opening in the drum instead of the whole canal causes less discomfort and seems to be more effective. A pledget of absorbent loosely placed against the drum before inflation or catheterizatnon tends to prevent the fluid from being drawn back into the tympanum.

Prognosis: The outlook depends upon the duration — whether days or months, the consistency of the secretion and the form of treatment instituted. As a general rule the earlier these cases are seen the better the prognosis and the less fluid present the quicker it tends to disappear.

When the symptoms have been present for only a few days, simple inflations clear up the vast majority of cases. On the other hand, if fluid has remained for months or years, often persistent treatment over a long period is required. In obstinate cases the drum may have to be opened many times before the condition finally clears.

Any associated nasopharyngitis should, of course, receive early attention. In children adenoidectomy is often indicated, and occasionally in adults the removal of enlarged posterior tips of the inferior turbinates is necessary.

Persistent nasal obstruction should be corrected and hypertrophied lymphoid tissue or enlarged lateral bands treated. After any of these operations it is wise to wait at least two weeks before inflating the tubes. Allergic factors must be attended to and the possibility of endocrine disturbances kept in mind.

Treatment: According to the late Prof. Alexander, who was a contemporary of Adam Politzer, attention should first be directed to the nose. Any infection in the upper respiratory tract, particularly, the sinuses, should be corrected. In treating the affected ear air inflations are carried out. When patients are seen early simple politzerization is usually sufficient, but in old chronic cases often the catheter is necessary. Usually air inflation lasts only a few days, so this should be repeated every second or third day while the nose is treated at the same time by some vasoconstrictor. Often a change of atmosphere helps, such as the dry air of the mountains or perhaps the seashore. Sometimes allowing a patient to use the Politzer bulb himself, providing there is no acute coryza present, will hasten his recovery. Alexander frequently carried out this procedure for a month before sending a patient away. Of course, in these days this is not always practical.

Air inflation acts best when the fluid is serous and is not nearly so effective when heavy mucus is present. If the head is bent forward and tilted so that the affected ear is uppermost, inflation tends to allow the fluid to drain away through the tube more easily. In chronic cases the results are better if the patient sucks in air while inflation is carried out. Sometimes during inflation patients complain of marked dizziness. This is due to increased pressure against the round window. Alexander insisted that the catheter be reserved for chronic cases as the mucous membrane has already been injured.

When the hearing is not improved after the successful use of the Politzer bag it is wise to incise the drum. There is a marked difference of opinion as to where the incision should be made. Some writers prefer the posterior inferior quadrant as it is further away from the inner tympanic wall.

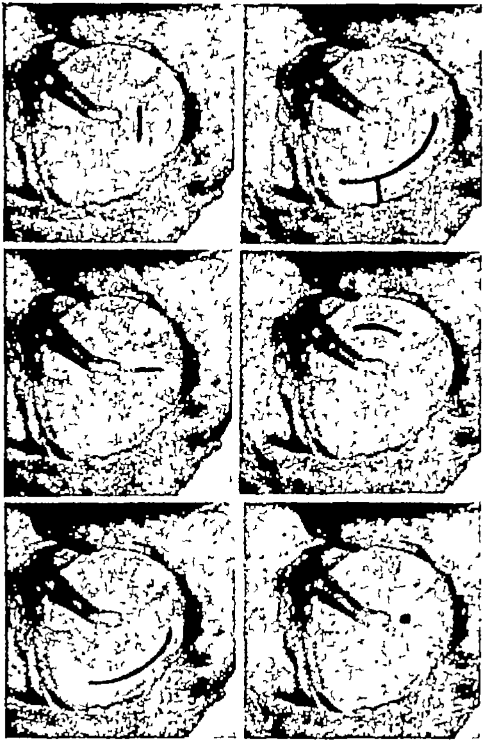

Fig 1

Others make a vertical incision just below the umbo. Still others suggest a horizontal incision below the end of the handle of the malleus, so that the floor of the external meatus is not injured. A favorite site for those who use a large hypodermic needle is just below the umbo at the six o'clock position.

In general it is well to remember that if there is pressure behind the drum the opening should be made in the most bulged part. At times it is necessary to make a second opening, especially if the secretion is of the heavy mucoid type. It has been suggested in recent years that this counter-opening be made in the upper anterior quadrant.

In the opinion of the author the initial incision should be made through the light reflex, *i.e.*, in the anterior inferior quadrant. The radiating fibres are the strongest in this area and as a result they tend to keep the incision open longer.

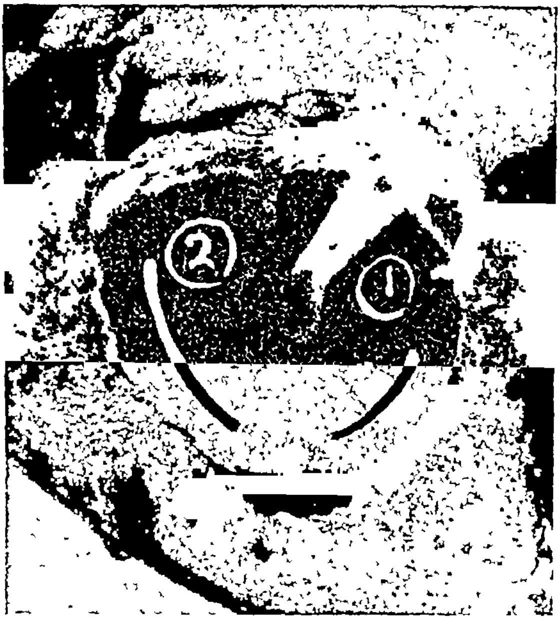

Fig. 2.

If myringotomy is performed anywhere else the opening usually closes within 24 to 36 hours. It is also inadvisable to make a large horseshoe incision as there is liable to be marked retraction and scarring.

The tough radiating fibres in the lower anterior quadrant have nothing to do with the cone of light. It is just a coincidence that they happen to be present in this region. After paracentesis air inflation should be carried out and spot suction. If no secretion appears and the hearing is not improved a second incision is recommended at once. This is made posteriorly, in the usual site for purulent otitis media. Often heavy mucus secretion becomes walled off, and this counter-opening is advisable.

Again after myringotomy repeated air inflations are often necessary, followed at times by spot suction or rarefaction by means of a Siegle's speculum.

When treated in this way over one-third (nearly 35 per cent) of cases of secretory catarrh clear up entirely without any after effects or repeated myringotomies. As children are commonly affected this is important. Occasionally repeated paracenteses are necessary.

Myringotomy should be performed without anesthesia and without swabbing the external canal with alcohol. Using a sterile paracentesis knife there is little danger of infection, and the discomfort can be compared to the momentary twinge of a hypodermic needle in the skin. On the other hand, a pledget of absorbent moistened in Bonain's solution (menthol, carbolic and cocaine in equal parts) placed against the drum in apprehensive patients does not seem to increase the chance of infection.

When indicated, myringotomy affords great relief, but this alone is not sufficient, unless contributing factors are corrected. Although the details and sites of paracenteses have been described, we should always bear in mind that almost 75 per cent of cases of secretory catarrh respond to conservative treatment. Relatively few in comparison require myringotomy in order to remove secretion from the tympanum.

SUMMARY.

A number of past and present day views on effusion into the tympanum have been enumerated. Although known and carefully described nearly 100 years ago, attention has been

In general it is well to remember that if there is pressure behind the drum the opening should be made in the most bulged part. At times it is necessary to make a second opening, especially if the secretion is of the heavy mucoid type. It has been suggested in recent years that this counter-opening be made in the upper anterior quadrant.

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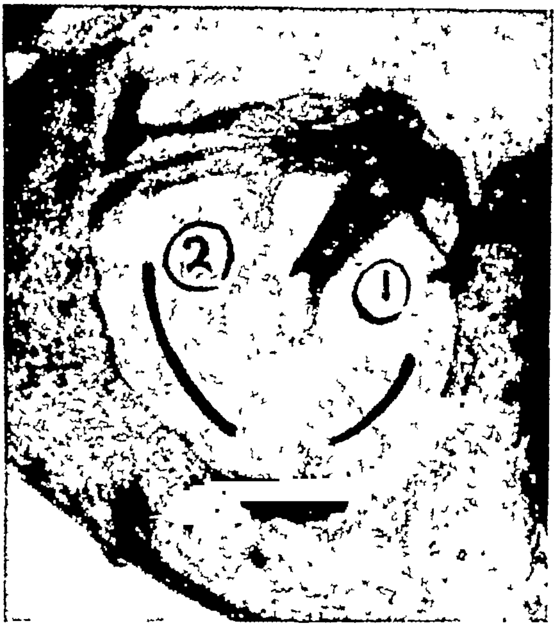

Fig 2

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SARCOMA OF THE NOSE AND SINUSES.*

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The American public has become cancer conscious. With the numerous articles appearing in current digests, newspapers, and women's magazines, the physician sometimes is hard put to keep ahead of his patients in the latest news on cancer detection and treatment. This is a desirable state of affairs, and now more than ever before, it behooves every specialist to have a wide and detailed knowledge of all malignancies that could occur in his field of practice.

Sarcoma arising in the nose or paranasal sinuses is a rare entity, but since two cases have occurred in our practice within a relatively short time, I considered the condition worth some special investigation, especially since both patients had been treated by other physicians who did not suspect malignancy.

History. Sarcoma of the nose and sinuses has been known and recognized for many years. Bosworth,[1] in 1889, published a book, "Diseases of the Nose and Throat," which included a chapter on sarcoma of the nasal passages. He collected 41 cases, about half of which were said to arise from the nasal septum. Most of these had not been examined microscopically.

The first published description of sarcoma of the nasal septum was a case report by Paletto, in 1820, and the first case which included an adequate microscopic study was reported by Mason, in 1875, according to R. H. Johnson, who

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reviewed the history of this lesion in 1904.[2] Clark (1898),[3] Baker (1899),[4] and Craig (1901)[5] also contributed early reports.

Etiology. The causes of sarcoma arising in the nose and sinuses are as obscure as those of most neoplasms elsewhere. In the literature no definite etiological factor appeared in most cases. In no case was trauma, chronic irritation or infection suspected as a probable cause. Irradiation, however, had a causative rôle in five instances.

Osteogenic sarcomas have been produced experimentally by means of X-rays and radium. Also cases of bone sarcoma have been reported after beta and gamma radiation. Wolfe and Platt[6] reported two cases of sarcoma occurring in the nasal bones in the region of the glabella. Both patients had received irradiation for squamous cell carcinoma of the skin in this area 15 and 20 years previously. Diagnosis of sarcoma was confirmed by microscopic sections of the tumor in each instance. They report a case published in the German literature by H. Hase, of a 12-year-old boy who developed sarcoma of the nasal bones. This child had had a scar over the nose, treated by X-ray irradiation at two years of age. They found 60 cases of widespread sarcomas reported in the literature up to 1948, where osseous sarcoma could probably be attributed to the effects of Roentgen ray and radium therapy.

W. G. Cahan[7] reported 11 cases of sarcoma developing after irradiation for some other cause. Two cases involved the sinuses. One was in a 26-year-old male who developed a sarcoma of the antrum 13 years after irradiation for a cloudy left antrum. Radical surgery was done and a three-year cure resulted. The second case was an osteogenic sarcoma of the ethmoid labyrinth which first appeared five years after irradiation for retinoblastoma in infancy. The tumor was radically excised; but it recurred, and the child died.

These cases should again remind us of the inherent dangers in radiation therapy. Its use must be reserved for potentially serious situations, and complications of treatment must be watched for constantly.

The so-called "cancer age" is not an important factor in sarcoma of the nose and sinuses. Many of the reported cases have been in children, and Pancoast[8] believes the disease to affect young people more often than the middle-aged; however, there has been only one case of sarcoma of the antrum diagnosed in approximately 90,000 admissions to Children's Hospital in Milwaukee in the last 26 years. This occurred in 1939 in a 10-year-old boy who died in spite of X-ray therapy. It is the impression of most authors that malignancy of the nose and sinuses in childhood is more rapidly growing and the prognosis is worse than when it occurs in later years.

Diagnosis. There are no typical symptoms of sarcoma, but certain findings may be suggestive. Some authors state that these tumors are slower growing with less tendency to ulceration than the carcinomas, and they present more distinct expansive growth with extension by contiguity, and symptoms due to pressure, rather than invasion and destruction as with the carcinomas. Nasal obstruction and pain are the two most frequent complaints. Wille,[9] reviewing 220 cases of malignancy at the Norwegian Radium Hospital, lists these presenting symptoms in order of frequency:

Nasal stenosis	41	Canthal swelling	8
Jaw and toothache	*38*	*Anesthesia of N V*	*6*
Pain in eye and temple	32	Loose teeth	4
Swelling of cheek	27	Impaired vision	3
Nasal discharge	21	Enlarged cervical glands	2
Alveolar tumor	12	Exophthalmos	2
Epistaxis	11		

X-ray examination is helpful in the diagnosis, and Pancoast[8] believes that sarcoma of the antrum can usually be diagnosed early by Roentgenograms. Homogeneous clouding of the antrum is present, but the appearance is not so dense as the clouded sinus due to polyps or thickened membranes with retained secretion. The original antral lesion is usually in the region of the teeth with the bone quite thin in that region. Alterations in alignment of the teeth due to encroachment by the lesion in the sinus is a later finding, and finally expansion and thinning of the walls of the sinus to a marked degree as the lesion increases in size. The bony outline of the sinus may disappear altogether. Fibroma is the only tumor which simulates sarcoma. These Roentgen findings confirm the clinical impression that the sarcomas are expansive rather than invasive growths. The contrasting X-ray picture of carcinoma of the antrum is one of erostion and destruction of the bony walls. The bone has a speckled or mottled appearance as though it was the seat of bone disease or invasion by a new growth.

The chief Roentgen sign of sarcoma of the ethmoid area, too, is the expansion of involved structures rather than invasion. For this reason it is often difficult to differentiate primary sarcoma from mucocele.

Final diagnosis depends upon examination of biopsy material or upon frozen section examination at the time of operation.

Pathology. The pathology of sarcoma is difficult to describe because of the various elements which may go into its formation. The amount of fibrous, cartilaginous, myxomatous, and osseous tissue and degree of cellularity, all are variable. Ohngren,[10] reporting from Stockholm in 1933, found 22 sarcomas (or 15 per cent) among 129 cases of malignancies arising in the maxilloethmoidal region; these were of the following types:

Osteoblastic	5	*Spindle cell*	2
Round cell	3	Plasmocytoma	3
Polymorphous	3	Myxosarcoma	2
Melanosarcoma	3	Fibrosarcoma	1

Ringertz,[11] of Helsingfors, has done a complete study of pathological types of sarcoma. He found that 5 per cent of all nasal and paranasal sinus tumors were sarcomata; that of this number, 20 per cent were fibrosarcoma or spindle cell sarcoma. Next in frequency were the myxomas and myxochondrosarcomas, and lastly the osteogenic sarcomas. In his experience this last group was by far the most malignant.

Most pathologists agree that the diagnosis cannot always be made from the histological picture alone. The clinical behavior of the tumor must also be considered. A growth which from histological appearance alone might be called a chondroma would have to be considered sarcomatous if it was locally recurrent and invasive. Such situations arise not infrequently. Also, in recurring, these tumors often become more cellular and assume a more malignant character.

Treatment. The evolution of treatment of malignancies of the antrum can be traced in a series of papers by G. B. New.[12-15] In reviewing the literature from 1926 he discovered that treatment by surgical excision had been generally unsuccessful and that irradiation alone had likewise produced poor results. In 1920,[12] New advocated eradication of the tumor by the actual cautery and implantation of radium into the marginal tissue. Later experiences led to the use of electrocautery instead of open cautery with radium. By 1926,[13] New had collected 97 cases of antral malignancy, 30 per cent of which were apparent cures. Half of these favorable cases were sarcomas. He reported further results in 1935[14] and 1938.[15] About 25 per cent of the total cases were sarcoma, and in these the results were particularly favorable — as high as 70 per cent five-year cures in one series.

It was pointed out by both Ohngren[10] and Holmgren,[10] who also favor this combined electrocautery-irradiation technique, that sealing of lymph and blood channels by the current will help prevent extension and recurrence. With ordinary surgical procedures there is more blood loss and more likelihood of spreading tumor cells to uncontaminated fields. Ohngren[16] reports 35 per cent five-year cures of maxillary sinus tumors but does not differentiate the types.

I. Szpunar,[17] of the Cracow University Clinics, uses electrosurgery as an adjunct to X-ray therapy rather than the reverse but emphasizes its importance in facilitating drainage, lessening infection, and promoting healing. He discusses 51 cases of malignant tumors of the maxillary sinus, of which five were sarcoma. He also mentions the use of nitrogen mustards in far advanced cases, feeling that they alleviate pain and temporarily improve the patient's general condition.

Each case must, of course, be individualized. If the sarcoma is early, readily accessible, and of low grade malignancy, electrosurgical excision is enough. Exact extent of the malignant tissue is, however, often difficult to determine; and if there is some doubt that excision is complete, prognosis can be improved if radium is implanted at the time of surgery.

If frozen section or biopsy shows a highly cellular tumor, some form of irradiation is mandatory in combination with electrosurgery.

When there is evidence of metastases, prognosis is grave and surgery useless, but X-ray irradiation will frequently reduce the size of the tumor and alleviate pain temporarily.

New[15] is of the opinion that the patient with a high grade malignancy has a better chance of primary cure by electrosurgery plus radium implants than one with a low grade tumor where radiosensitivity is low and surgery alone must be depended upon.

CASE REORTS.

Case 1: Mrs. A. G., age 40, was referred to us by an ophthalmologist in January, 1946. The patient had complained of a dull aching pain over the right frontal sinus and right eye, and a feeling of fullness in this region since October, 1945. Several severe, right-sided nose bleeds had also occurred. The doctor had recently operated for what he thought was a chronically infected lacrimal sac on the right, but he did not complete the operation because of hemorrhage. Positive physical findings were limited to the head. A hickory nut sized swelling was seen above and medial to the right inner canthus, and there was profuse tearing of the right eye. Vision was normal. The ears, throat, nasopharynx and larynx were normal. Intranasally there was some granulation-like tissue in the right middle meatus. This tissue bled easily on slight manipulation. No pus was seen in the nose, but the tissue present suggested some trouble in the sinuses on the right side.

X-ray changes were minimal considering the size of the external swelling. There was an asymmetry of the frontal sinuses particularly in the region of the frontoethmoid cells. There was lack of trabecular formation on the right side, but no actual bone destruction nor tumor formation could be seen (see Fig. 1, Case 1).

A tentative diagnosis of mucocele was made.

A right intranasal ethmoidectomy was attempted in April, 1946. Tissue was obtained for microscopic examination, but operation could not be completed because of hemorrhage. The pathologist reported inflammatory tissue. We planned to complete the operation through an external approach, but the patient found it necessary to leave the hospital because of a family emergency. We were able to see her occasionally in the office, and each time she was urged to have the operative work completed. Her husband remained an invalid for many months, and the patient felt she could not re-enter the hospital. The external swelling increased slowly and the right eye began to protrude before we were able to hospitalize her again in February, 1948. The intranasal picture had also changed. The right lateral wall of the nose was pushed against the septum, making it impossible to visualize the right middle meatus. X-rays at this time showed definite change. The anterior ethmoids, the anterior portion of the frontal bone in the region of the superior and inner aspect of the right orbit, and the septum between the two frontal sinuses could now no longer be demonstrated. There was a 1 cm. soft tissue shadow projecting from the right into the left frontal sinus (see Fig. 2, Caes 1).

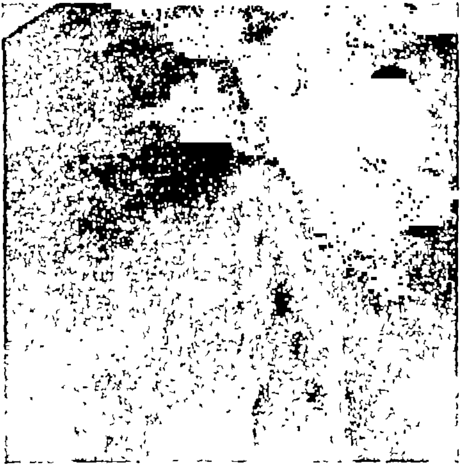

Fig 1. Case 1. Waters projection taken February, 1946, showing some asymmetry of the frontal sinuses and lack of trabecular formation on the right, in the frontoethmoid region. No tumor demonstrated.

Right external frontoethmoidectomy was done in February, 1948. Immediately beneath the skin and subcutaneous tissue, a very thin shell of bone presented itself. There were several areas where bone was absent, and sinus mucosa was seen. In removing bone for better exposure, we found what appeared to be a cyst wall. Practically all of the floor of the frontal sinus and the medial wall of the orbit were missing. The wall of the cyst varied from 1 to 3 or 4 mm. in thickness. A small amount of thick mucus was found within the cyst Bleeding was profuse throughout the operation, mostly from the eroded medial wall of the orbit. A 500 cc. whole blood transfusion was given during operation. A dehiscence was found in the septum between the frontal sinuses, and a bit of tissue protruded through the opening from the right into the left frontal This tissue was removed, and the remainder of the frontal septum was excised. The mucosa of the left frontal was normal.

Fig 2 Case 1 Granger 23° projection taken February, 1948 Here definite bone destruction in the region of the right anterior ethmoids and right orbit is seen The bit of tissue, protruding from the right into the left frontal sinus found at operation, is clearly visible

When operation was completed, all remnants of cyst wall, the entire floor of the right frontal sinus, the septum between the two frontals, and the lateral wall of the nose had all been removed This left a large cavity which required tight vaseline gauze packing because of hemorrhage The external incision was closed with black silk Frozen section of the tissue was not done because of the benign appearance of the cyst and its contents. Fifty thousand units of aqueous penicillin were administered every three hours, and the patient was given three 500 cc transfusions of whole blood on successive days because of continued bleeding. The postoperative course was otherwise uneventful Packing was removed in stages over several days.

Microscopic examination revealed a cellular tumor, the cells of which closely resembled those of cartilage. The lesion was not encapsulated and appeared locally invasive Review of microscopic sections prepared in 1946, together with consideration of the course of the lesion, strongly supported the interpretation that this represented a neoplastic rather than an inflammatory lesion Diagnosis: chondrosarcoma, low grade, ethmoid and frontal sinuses (see Fig 3, Case 1) After learning the pathologic diagnosis, some form of irradiation was considered The patient was three days postoperative at the time, and with the bleeding the use of radium intranasally was not feasible External irradiation might cause breakdown of the wound so that this method was rejected From the pathological sections, we felt that this tumor was probably quite radioresistant and in view of the fact that we were reasonably sure all diseased tissue had been removed, we decided against irradiation.

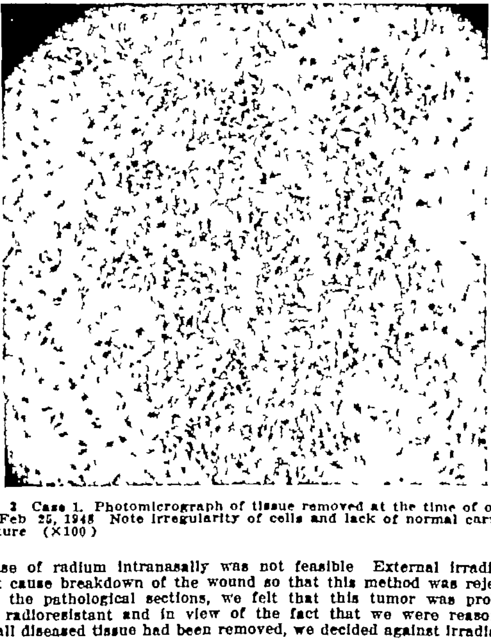

Fig 2 Case 1. Photomicrograph of tissue removed at the time of operation Feb 25, 1948 Note irregularity of cells and lack of normal cartilage structure (×100)

The external wound healed nicely, and over a period of time the intranasal cavity epithelialized completely

It was discovered on the third postoperative day that vision in the right eye was reduced to light perception only. An ophthalmologist felt that this was the result of our firm packing pressing either directly on the optic nerve, or on nutrient blood vessels to the eye—certainly an unexpected complication.

The patient has been observed in the office repeatedly. She was last seen in the office in March, 1950. There was no external swelling. Intranasally the operative cavity was completely clean and free of any recurrence. Vision remained poor in the right eye.

Comment. The loss of vision in one eye was a most unfortunate complication. Perhaps it could have been avoided by removing the packing sooner. A better method in theory would have been the use of electrocautery which might have made the tight packing unnecessary. Actually, we were following a line of cleavage in dissecting out the cyst and sharp dissection was necessary.

In retrospect, we think this to have been the course of the disease: A slowly developing mucocele of the right anterior ethmoids had most likely been present for several years without producing symptoms. In October, 1945, when epistaxis began, the sarcoma within the cystic mucocele probably had broken through into the nose. This occurred because the tumor was exposed in the middle meatus to any infection in the nose. External swelling was also observed at this time.

When the right external frontoethmoidectomy was done, the cyst wall was intact except in the middle meatus. This has led us to believe that this case represents the development of a sarcoma within a mucocele.

Case 2: We first saw the patient, E. V. P., age 50, July 25, 1947. She complained of nasal obstruction for which she had been under observation by an out-of-town doctor for one year. Local treatment by packing, the use of nose drops, and steam inhalations had failed to relieve her symptoms. An allergist, who had recently been consulted, felt that the obstruction was not on an allergic basis and referred the patient to us. There was no history of trauma or infection to account for the obstruction.

Ear, nose and throat examination was negative except for the nose. Externally the nose was normal. The nasal mucosa was pale, somewhat boggy and suggestive of allergy, and there was an excess of clear mucus in the anterior portion of both nasal chambers. Further back in the nose there was a symmetrical, smooth, bilateral swelling of the nasal septum, covered completely by intact mucosa. There were no areas of roughness, erosion or bleeding. The swelling began in the midportion of the septum, extending upward from the level of the superior border of the inferior turbinates, and backward to the posterior choanae. It was not possible to get an adequate view of either middle meatus or the middle turbinates. The inferior portion of the septum was uninvolved. On palpation the swelling was smooth and hard. The frontals were clear and the antra hazy on transillumination. The nasopharynx was normal X rays revealed a cystic swelling of the nasal septum without bone destruction. There was some clouding of the left antrum (see Fig. 4, Case 2)

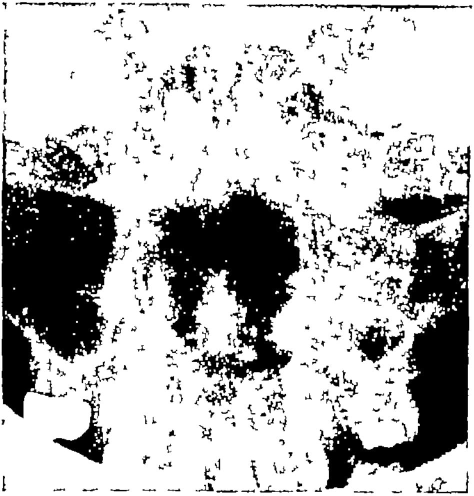

Fig 4 Case 2 Granger 107° projection taken July 1947, demonstrates the cystic swelling of the nasal septum. Some clouding of the left antrum is also noted.

Because of the length of time the symptoms had been present and its benign clinical and radiological appearance our working diagnosis was a benign bone cyst of the septum, although we were unable to find any such condition described in otolaryngological or pathological texts The possibility of a primary malignancy of the septum could not be excluded. Routine laboratory studies were normal and the patient had no fever

It was decided to approach the lesion surgically by means of a standard submucous resection of the septum and this was done on July 30 Anesthesia consisted of cocaine crystals moistened with 1/1 000 adrenalin applied topically After separating the mucoperichondrial flaps in the usual manner and reaching the region of the swelling a semisolid material was encountered The perpendicular plate of the ethmoid was missing. The tissue was relatively avascular but looked a great deal like malignant tissue having the granular tapioca like appearance of mixed salivary gland tumor or chondroma. Tissue was sent to the laboratory for frozen section. *The pathologist s diagnosis was a highly cellular,* malignant growth; probably a sarcoma. All visible tumor was removed up to within 1 to 2 mm. of the cribriform plate. The tumor extended to the posterior choanae and had eroded, probably by pressure, the entire anterior wall of both sphenoids. It was necessary to curette laterally from the anterior wall toward the greater wing of the sphenoid on both sides in order to remove tumor tissue, but no tumor was seen within either sphenoid sinus. At completion of operation the mucosal flaps were still intact and normal on the external surface, but somewhat roughened on the medial side. All visible tumor had been removed.

Gauze packing was placed in the nose and left for 24 hours. Penicillin was given for one week. There were no postoperative complications.

Later, paraffin pathological sections (see Fig. 5, Case 2) were reported. Microscopic examination revealed the presence of a malignant tumor composed of cartilage, which in some areas was undergoing myxomatous degeneration. The tumor was invading and destroying the nasal septal cartilage and bone. Paraffin sections were not as highly cellular as the frozen section preparation, which would suggest that the tumor was not growing as rapidly as previously thought. The final diagnosis was chondrosarcoma of the nasal septum.

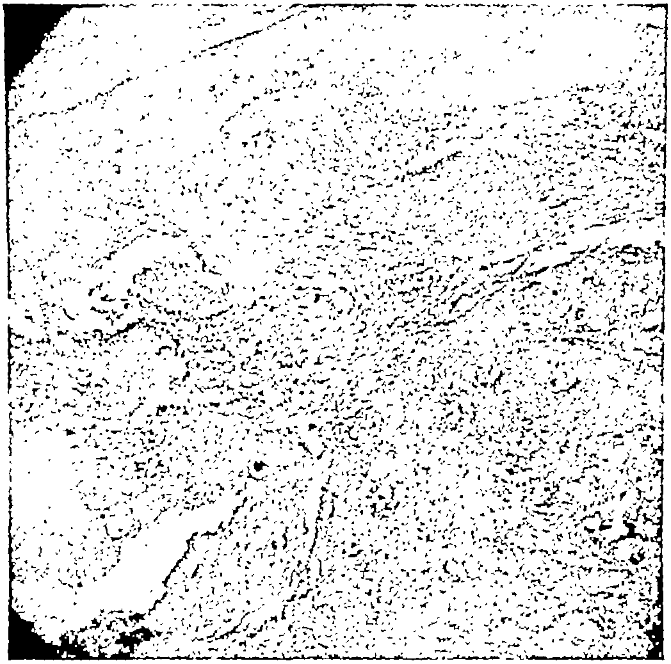

Fig. 5. Case 2. Photomicrograph showing section of tumor removed surgically, July 30, 1947. Atypical cartilage cells and myxomatous stroma are present. Also the tumor is seen invading and destroying bone and cartilage. (×100.)

Since we could not be sure that our excision was adequate, we decided in consultation with the Roentgenologist to use some type of irradiation, in spite of the fact that this type of malignancy is reportedly not radiosensitive. On the ninth day after operation, 145 mg. of radium were put into the nose and packed as high as possible with vaseline impregnated gauze strips. It was left in place 12 hours, delivering 1,740 mg. hours. The only immediate postirradiation reaction was some dryness in the nose. Dryness and crusting of the nasopharynx, particularly on the left side, has been a problem. We have had the patient use plain olive oil in the nose to lubricate the nasopharynx, and she has also irrigated the nose with saline to help remove crusts. On three or four occasions there has been a little serous fluid in the left middle ear, which we have been able to relieve by inflations of the left Eustachian tube.

The patient has complained of occasional pain in the left side of the face and neck. No organic basis has been found to explain it, and we hope it is only a later effect of the irradiation therapy.

Ten months after irradiation had been given, a half dime sized ulceration was observed on the posterior pharyngeal wall. Biopsy of the margin of the ulcer showed only inflammatory tissue. It was decided that the ulcer was probably due to the radium, and it subsequently healed. The patient was last seen in August, 1950, at which time no recurrence of the tumor could be seen.

Comment. Primary chondroma of the nasal septum has been reported and is apparently not uncommon, but we have been unable to find a single case of chondrosarcoma similar to this case. Its site of origin in the septum cannot be questioned because we were fortunate enough to see the patient before it had spread to adjacent tissues. A cure cannot be claimed, but as no recurrence has occurred up to this time, we feel reasonably certain that none will.

SUMMARY AND CONCLUSIONS.

1. Sarcoma of the nose and sinuses is rare.

2. These tumors occur more often in childhood and adolescence than carcinomas. In most of the cases in the literature, no etiologic factors are evident, but in at least five cases sarcoma was preceded by irradiation to the region.

3. Sarcoma of the nose and sinuses grows by expansion rather than invasion, and has some characteristic Roentgen findings.

4. Exact pathological diagnosis is often difficult due to the various connective tissue elements which can comprise the tumor.

5. Treatment by electrosurgical excision augmented by radium therapy gives the best results in most cases.

6. Two cases are reported. The first case is unusual because a chondrosarcoma apparently developed within a mucocele. The second case is a primary chondrosarcoma of the nasal septum, a lesion which, so far as we know, has not previously been reported in medical literature.

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ENDOTRACHEAL INHALATION ANESTHESIA.

SPECIAL REFERENCE TO POSTOPERATIVE REACTIONS AND SUGGESTIONS FOR THEIR ELIMINATION.*

PALUEL J. FLAGG, M.D. (by invitation),
New York, N. Y.

Last March I wrote each member of the American Laryngological, Rhinological and Otological Society, Inc., requesting his experience and judgment respecting the incidence of trauma in endotracheal intubation for anesthesia. In reply to the 514 inquiries directed to the total membership, including ophthalmologists, otologists and laryngologists, 154 responses were received. This total necessarily represents much more than the 30 per cent implied; in fact, it may be assumed that the majority of those interested in laryngology were kind enough to reply. These responses were assembled as a survey and a mimeographed copy was mailed to each respondee. This survey now appears in the April issue of the *Archives of Otolaryngology* released last week. My purpose today is to state more fully why this inquiry seemed necessary in the first place; second, to give you some of the reactions to it; third, to note a drift which is taking place in the whole field of anesthesia; and finally, to emphasize the details of the technique which, in my hands, has been almost entirely free from regrets.

May I first enlarge somewhat on the background upon which my use of this method was predicated.

It is common practice in medicine for a specialist in one field to turn to that of another when problems overlap. It is

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*Read at the Fifty-fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 8, 1951.

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most unusual for men in one field to attempt to establish through experience findings referable to another; and yet, in the field of endotracheal anesthesia it would seem that the clinical reaction to instruments and technique has been employed to establish rules and regulations for present practice, rather than the experience of the laryngologist gained through an intimate acquaintance with physiological tissue reactions to instrumentation. How often, for example, does the laryngologist appear in the introduction of a new endotracheal tube or technique,—and yet, should not the use and safety of such instruments turn upon laryngoscopic experience rather than anesthetic trial and error?

Wherever endoscopy is used in other fields, the physician employing it lives with his patients through diagnosis, operation, immediate recovery and ultimate after effects.

In my initial use of endotracheal intubation, I turned to Chevalier Jackson. A carbon of a letter, dated Nov. 29, 1926, is before me. I asked Dr. Jackson the following questions: What is the usual reaction to intubation? What is the maximum outside diameter of the bronchoscope for the adult and the child? Would a flexible tube give less reaction? How long may the tube be left in the larynx without trauma? Will application of lubricants reduce the effect of intubation? Would a large size tube be less likely to traumatize than a smaller tube?

Jackson's replies were precise and afforded a safe base for action. Following further correspondence and trials, Jackson wrote me three months later (under date of Feb. 23): "I would suggest that you make a preliminary publication at the earliest possible moment." I replied: "No preliminary observation of my own would have any value. This would be merely speculation. You alone can pass judgment on the soundness of this method. If you say that such and such a technique, or a tube of such a size is correct, that is the end of it. Later on, in a further report of the method, my opinion based upon case experience will be of value."

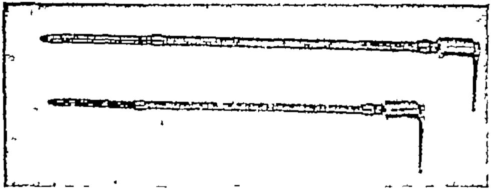
For 10 years I did endotracheal anesthesia with the tubes recommended by Jackson, under every environmental condition, in large hospitals, in small hospitals, in sanitariums and in homes, at leisure and under pressure. The results paralleled bronchoscopy. They were practically without regrets. New tubes, new laryngoscopes made their appearance; blind intranasal intubation became popular; carbon dioxide absorption technique, ethylene, cyclopropane followed each other. Through the daily contacts with which we are all familiar were voiced disturbing reports; seven granulomas in the practice of one laryngologist; sloughing of tracheal mucosa from balloon pressure followed by death; from another: stories of long struggles to intubate; an emergency call to assist in the intubation of a patient; apnoeic from oversedation; resistant to an attempt to intubate an oversized tube but offering no resistance or difficulty to the 10 mm. tube in our use; and then there was the threat behind it all: the threat that all endotracheal intubation might be condemned, including my own fortunate experience, unless the reasons for these accidents could be discovered. It seemed quite clear that the policy of trial and error to determine tolerance of the glottis, the attempt to accommodate the size of the endotracheal tube to the requirements of anesthesia machines was proving disastrous. A new and more complicated problem, one which I trust will not be judged presumptuous, was taking shape; *i.e.*, to investigate the rumors of trauma, to bring up to date the original postulates of Jackson, and to integrate these results with my personal experience for the benefit of the patient and for the protection of the field.

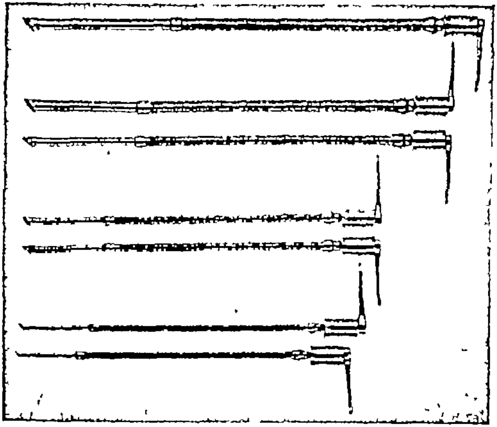
In 1926, I turned to Chevalier Jackson for advice. Through the last year I have come to you. You have responded most generously. You have reported pathology which has come to your attention following endotracheal anesthesia. You have confirmed the views Jackson stated to me 25 years ago.

May I now refer briefly to the questions placed, and to the *resulting summary* which appears in the April issue of the *Archives of Otolaryngology.*

Trauma Following Bronchoscopy.

In routine laryngoscopy and intubation for bronchoscopy 98 per cent expect little or no reaction.





Consultations for Trauma Following Anesthesia.

Ninety-nine respondees, or 64 per cent, were called in for postoperative trauma following endotracheal intubation for anesthesia.

From Connecticut: Today most endotracheal intubations or anesthesia are done using pentothol as the basic anesthesia. Most anesthetists simply do not take time to anesthetize the throat and larynx properly with local anesthesia. When spasm occurs, as it frequently does, then trauma is produced in the hurry and excitement. The anesthetists know they will never have spasm to contend with if I fix the patient first.

THE EFFECT OF LONG INTUBATION IS REFERRED TO.

Louisiana: It is my opinion that bronchoscopy is less traumatic than endotracheal intubation for anesthesia due to the fact that bronchoscopy requires only a few minutes during which the bronchoscope is in contact with the vocal cords. On the other hand, the intubation tube frequently remains in contact with the vocal cords as long as several hours and this constant pressure, particularly if the patient is not under deep anesthesia, produces considerable trauma of the cords.

Massachusetts: I have often wondered if the prolonged presence of the tube in the larynx has any influence in the etiology of the traumatic granuloma which sometimes occur. On the other hand, I recall that prolonged intubation of the larynx for laryngeal diphtheria in the old days rarely caused any such trouble.

OBJECTIONS TO BLIND INTUBATION ARE REPEATED.

Pennsylvania: I have encountered a number of cases where the larynx was traumatized, and I have noted many more cases of traumatism on the nasal passages, due unquestionably to the blind intubation through the nose, of which I heartily and thoroughly disapprove.

Chicago: Certainly blind bouginage as well as the use of oversized tubes and intralaryngeal pressure are all to be condemned.

New York: I feel that this is a very important stand for you to take as I have seen a great many cases with hoarse voice, over months and months, and also cases where nose bleed developed following intratracheal anesthesia. Most of the cases I have seen have been cases that were induced by the blind method of nasal catheter.

THE EFFECT OF LOCAL ANESTHESIA LUBRICANTS ON THE ENDOTUBE IS POINTED OUT.

California: I would like to add that the worst immediate reactions to intubation that I have seen were traceable to the use of anesthetic lubricants, nupercaine and intracaine, causing contact dermatitis, so that it is not surprising that an occasional patient will show a pseudomembranous reaction in the larynx and subglottic region. I have not seen this reaction to intubation except where such agents were used.

THE FOLLOWING COMMENTS STRESS ACCIDENTS.

New York: Let me mention one case which I blame in part on endotracheal anesthesia. This was in a boy who had been run over and had a ruptured spleen. There was no evidence of injury to his chest. In doing the spleenectomy, endotracheal anesthesia was employed. He had respiratory difficulty on the table which prevented removal of the tube after the operation was completed. Two hours later he was dead, and at autopsy it was discovered that he had a massive tension pneumothorax from a tiny laceration in the monor fissure of his right lung. I accepted most of the blame for this, because I should have recognized that there was a tension pneumothorax; but, on the other hand, if the anesthetist had not been pumping anesthetic gas into him by squeezing on the bag, the boy would not have had the tension.

I know of three deaths due to improper intubation and a fourth was prevented only by wide incisions in the neck the day after operation. Two of these were cases of my own.

New York: I had to stand by for hours while a case that was in great respiratory distress was being considered for a tracheotomy. The patient had endotracheal anesthesia for a thyroidectomy. Both cords were immobile and in the midline, but the vocal processes moved on attempted phonation, which ruled out a paralysis of the recurrent laryngeal on both sides. The consensus of opinion in this case was that the larynx was damaged by trauma of endotracheal anesthesia.

I have attempted to investigate rumors of trauma following endotracheal anesthesia. The recommendations made to me by Jackson 25 years ago are almost identical with those which you have stated and which now appear in the April issue of the *Archives.*

Your responses indicate that laryngoscopy and intubation in bronchoscopy is relatively nontraumatic, that laryngoscopy and intubation for anesthesia is markedly so.

The first step to take in establishing the etiological factors responsible for this reaction would seem to be to note the variances in the methods.

The first difference is the intubation of an unconscious patient. The mechanical variances to be noted are larger tubes, blind intubation and the use of subglottic packs. If the reasons for the pathology reported are to be found in these variances, their removal should eliminate them.

While the survey offers no broad evidence to bear out the logic of this conclusion, it is a matter of revealing interest to reflect that by reason of conservatism or, possibly, lack of originality, the endotracheal tubes which I have used have never exceeded the outside diameter which you recommend. I do not use blind intubation. I do not employ subglottic packs; first, because I have found such tubes too bulky; and, second, I have found them unnecessary. My patients have always been adequately relaxed with ether. Postoperative trauma in my hands has not exceeded that which you report for routine bronchoscopy. As a matter of fact, it has been less than 2 per cent.

I now comment upon the matter of general anesthesia, the field obtainable under ether anesthesia, the instruments which I have used and, finally, what I believe to be the implications contained in the popularization of laryngoscopy and intubation in the relaxed patient.

May I point out what seems to me to be a major drift affecting the whole field of anesthesia? A realization of this drift will help to eliminate much of the mystery presently complicating anesthetic practice and postoperative reactions.

It seems clear that anesthesia has drifted from pneumatology, the use of gases, to pharmacology, the use of drugs, administered by mouth, rectum, hypodermic, vein and spinal canal. This drift is accompanied by the complications common to pharmacology. Dosages are irreversible, effects are often unpredictable and idiosyncrasies are not uncommon. In therapeutics, pharmacology anticipates these complications and guards administration by establishing a therapeutic index for each drug, a margin of safety between the effective medical dose and the lethal dose.

When, however, drugs are called upon to eliminate consciousness, muscle tone and reflex activity, to depress respiration to the vanishing point, what has become of the therapeutic index, the margin of safety? Furthermore, when one depressant drug is added to another, in order to produce a suitable surgical field, is not the result shrouded in mystery?

What is back of this drift? Is it not ease of administration, an agreeable induction and euphoria upon recovery skillfully integrated and attractively presented by pharmaceutical firms? The power of these motivating forces are great. An appeal to safety and controllability may induce the patient to tolerate gas therapy; it will not make it agreeable. The foregoing is not intended as a blanket condemnation of the substitution of pharmacology for pneumatology. It is an attempt to bring into perspective the limitations as well as the usefulness of these sciences to help the surgeon to anticipate the normal limits of control of gases and drugs. In the light of these facts, one may more easily appraise and assist present practice in anesthesia.

While pharmacological control, with preoperative sedation, morphine, scopalamine, pantopon or demoral, with loss of consciousness by intravenous pentothal-sodium, an agreeable induction is begun. This medication does not accomplish muscle relaxation or reflex control of the glottis. The field at this point resembles that of laryngoscopy and intubation under local anesthesia. To complete the preparation of the *field by the pharmacological approach, curare or syncurine* is used for relaxation, and topical pontocaine or cocaine is used for control of the glottic reflexes. The doses of these drugs and the individual's reaction vary. Increased control is secured at the cost of reducing the margin of safety. Pharmacological anesthesia is common practice. The skill required for its use is largely that of venapuncture and observation of the effects of the drugs used. Antidotes are singularly ineffective in unexpected reactions. Emergency measures for relief of laryngospasm and respiratory depression turn upon positive pressure, insufflation, through the endotracheal tube.

Anesthesia is maintained by virtue of the continued effect of preoperative sedation to which is added barbiturate hypnosis. Recovery is likely to be delayed and is occasionally complicated by the characteristic barbiturate respiratory depression which cannot be overcome by the use of carbon dioxide. As a rule, the patient awakens free from unpleasant effects and with an inclination to euphoria.

Pneumatological control of general anesthesia, carried out with gas-oxygen-ether, is, on the other hand, reversible in its effects; its effects are predictable to a high degree but prefaced by an induction which is sometimes unpleasant.

Maintenance is under an elastic control which leaves a wide margin of safety between adequate muscle relaxation, control of the glottic reflex and depression of the respiration. Where such anesthesia is induced without sedation, as is my practice, induction is more rapid, relaxation is more easily secured and recovery prompt. Patients recovering from ether anesthesia with endotracheal control, as a rule, have little nausea or vomiting. This is particularly true in operations about the head and neck. A brief period of recovery is followed by a prompt return of normal vigor. Where two or three children are anesthetized one after the other for squint operations, the first is usually sitting up in his crib when the third is returned to bed. With the exception of occasional preliminary avertin, the speaker prefers to use no preliminary medication. Ether usually provides a relaxed field in from eight to 12 minutes.

A word as to this relaxed field is in order. There is a sharp and most important difference between laryngoscopy and intubation of the conscious patient under local anesthesia and the patient who is limp from adequate general anesthesia or who is in the flaccid stage of asphyxia. In the first, there is the familiar resistance of muscle tone and a variable degree of reflex activity. In the second, exposure is no more difficult than is exposure by a tongue depressor or nasal or oral speculum. This difference is so real, and its importance in instrumentation is so great that the laryngologist who limits his work to exposure under local anesthesia should go out of his way to familiarize himself with the freedom of exposure in relaxation. A field which is suitably relaxed brings this instrumentation within the range of many who would otherwise be excluded as unskillful.

Given a relaxed field, in which the glottis may be exposed and intubated with ease and without haste, much of the objection which has been raised to the wide use of laryngoscopy from the trauma caused by tense muscle and reflex spasm, would disappear.

Would it not be well worthwhile for the laryngologist who is faced with anesthetic difficulties to laryngoscope and intubate his own patient, thereby eliminating the chief hazard to head and neck anesthesia?

Now a word regarding instruments: it will be found that a laryngoscope blade at least seven inches long, with a large light and an opening to the side will provide a better field of exposure and allow easier intubation than does the conventional laryngoscope.

The tube which I have used has been previously described in detail. (Reference will be found in the April issue of the *Archives*). A set of seven tubes are now available, beginning with 4 O. D. up to and including 10 mm. O. D. While some prefer to connect the endotracheal tube to a carbon dioxide absorption canister or to a rebreathing bag, I have found that what has been referred to as the "tin can" inhaler leaves little to be desired. Inspiration and expiration are entirely free. Negative and positive pressure, 2 or 3 mm., in the closed inhaler may not seriously inconvenience the patient, but complete freedom is preferable.

The purpose of endotracheal inhalation anesthesia is, first, to provide unobstructed respiration, in adequate volume; second, to prevent aspiration of foreign matter from the pharynx. Beecher has shown that the tubes which I have employed provide adequate ventilation (Henry K. Beecher, *Anesthesiology*, Vol. II, No. 6, 1950). Large tubes and tubes with subglottic balloons have been employed by many to reduce aspiration around the tube. I have found that a pharyngeal pack introduced over the top of the tongue deep into the pharynx prevents regurgitation of gastric contents into the hypopharynx and the draining into it of secretions from the mouth. The occasional seepage of fluid past the tube is immediately evident in the sound of the respirations through the tube. Such secretions are immediately removed by suction catheter.

One of the most important factors of safety during both induction and maintenance of endotracheal anesthesia is adequate suction. Its presence in the anesthetizing and operating room is mandatory.

At this point I should like to submit for your consideration a new tube which illustrates the principle of the Mosher lifesaver. A 5 and a 7 mm. endotracheal tube terminates in a tapered catheter-like tip. This tip is supplied with perforations, the total area of which is more than twice the area of the lumen of the tube. This new tube has a double purpose: first, it provides a means of entering a spastic glottis without trauma; second, it serves as a practice instrument for the student learning to intubate.

The need for this tube has been precipitated by the frequency and the danger of severe laryngospasm accompanied by respiratory depression reported during pentothal anesthesia. The ease and the frictionless entrance of this lubricated tube through a spastic glottis must be experienced by the operator in order to be appreciated. It is believed that this type of tube will serve a useful purpose in accidental asphyxia requiring intubation.

In conclusion, may I refer to the need of popularizing laryngoscopy and intubation in the relaxed, asphyxiated patient? Where one is about to die from respiratory obstruction and where the only possible relief is in the hands of rescue squads, it is becoming increasingly evident that at least one member of each squad should be oriented in the death zone of the respiratory tract, taught how to relieve pharyngeal obstruction and instructed in the simple technique of intubation of the completely limp patient.

To this end I have for four years given regular monthly courses of laryngoscopy and intubation for the treatment of acute asphyxial accidents, to pediatricians, obstetricians, hospital residents, nurse-technicians, and selected members of rescue squads.

Orientation of the field has been accomplished by the use of anesthetized dogs, anesthetized patients, and cadaver. The saving of many lives has resulted.

This course is given monthly and is completed in a Friday afternoon and Saturday morning session. The fifty-first class met last Friday and Saturday.

SUMMARY.

The importance of cooperation between the fields of laryngology and anesthesia is stressed. Pathology following endotracheal anesthesia is discussed. The present drift of anesthesia from pneumatology, the use of gases, to pharmacology, the use of drugs, with its implications, is stressed. The difference between laryngoscopy in the conscious patient un-

der local anesthesia and in the unconscious patient, relaxed by general anesthesia or asphyxia is noted. Instruments used by the writer are referred to, and a new lifesaver tube is demonstrated. A plea is made for the popularization of laryngoscopy and intubation in the relaxed patient with an obstructed airway. Courses of instruction for all those responsible for the care of the unconscious patient are offered.

FRONTAL SINUS:
PERMANENT DRAINAGE WITHOUT DEFORMITY.*

HARRY P. SCHENCK, M.D.,
Philadelphia, Pa.

While surgical approach to the frontal sinus may become necessary in the management of neoplasms, the removal of foreign bodies, or the reduction of fractures of the frontal bone, it is usually carried out because of the spread of infection to the bony walls or beyond the confines of the sinus.

Since the introduction of sulfonamides and antibiotic drugs, *conservative measures, together with chemotherapy, have controlled most acute infections of the frontal sinus, reducing* the incidence of complications and of chronicity of infection.

In spite of the advances in chemotherapy, there are occasional instances when extension of infection takes place to the bony walls, subperiosteal abscesses or draining, fistulous tracts appear, and meningitis, osteomyelitis or brain abscess become complicating factors. Neglect, delayed or inadequate therapy, the use of the wrong chemotherapeutic agent, inept intranasal intervention or the presence of structural defects probably account for most of these sequelae.

Chronic sinus infections are resistant to antibiotic therapy because of the difficulty with which the drugs penetrate abnormal tissues resulting from repeated or prolonged infection. In the case of the frontal sinus, *once irreversible tissue changes occur within the sinus and the nasofrontal ostium* reinfection and chronicity of infection are inevitable and complications common.

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*Read at the Fifty-fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., May 7, 1951.

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Regardless of the reasons for external surgical approach to the frontal sinus, the selected technique should accomplish the aim of the operation with the least residual deformity and scarring. Since the introduction of antibiotics, these objectives are usually attained by minimal sacrifice of sinus walls, the construction of a permanent drainage tract from the frontal sinus to the nasal cavity and immediate closure of the external wound. During the preantibiotic era, when wide removal of all diseased bone was imperative because of the lack of known controls against the spread of infection within bony structures, the operations of Riedel, Killian, Lothrop and Lynch were lifesaving measures. Today they are often no more effective than simpler procedures and entail needless sacrifice of functioning structures and membranes.

Osteomyelitis, with or without brain abscess, has rarely been cured by antibiotic therapy alone, but antibiotic therapy together with surgical drainage has effected some remarkable recoveries. Even the view that all necrotic bone must be removed before healing can occur may need revision. Although the antibiotics exhibit a peculiar ability to relieve the pain associated with osteomyelitis, their main value lies in the prevention of sequestration and in the arrest of progressive extension to contiguous osseous tissue. Their timely use in osteomyelitis secondary to sinus infections has not reduced the need for surgical drainage but it has reduced the extent of radical surgery. The question today is, "What constitutes adequate surgical drainage?"

After external entrance to the frontal sinus and the completion of the necessary intervention within it, an efficient drainage tract from the sinus to the nasal cavity must be maintained. Following either radical, deforming operations or more simple procedures, such a drainage tract is prepared by enlarging or reconstructing the nasofrontal approach. Because the subsequent constriction of the new channel is a serious matter, various methods have been employed to prevent it, either by the use of skin grafts in the frontal ostium and the nasofrontal channels, or by the introduction of tubes of tantalum, plastic or rubber.

The following simple technique has been employed for many years in the absence of bone infection, but since the introduction of the antibiotics, it has been used to the exclusion of more radical operations excepting under unusual circumstances.

TECHNIQUE.

Twelve hours prior to operation, the site of the incision is outlined by a linear application of 25 per cent silver nitrate solution which follows the midline of the eyebrow. The hair-bearing surface is later shaved to insure surgical asepsis, and the predictions of subsequent objectionable effects of shaving have not been borne out in more than 100 operations.

Endotracheal anesthesia is preferable, but in the absence of serious infection, the operation can be readily carried out with local anesthesia. The incision through the skin is continued through the periosteum to the bone and its length restricted to the exact requirements of the operation. Digital pressure by an assistant controls the profuse hemorrhage from the branches of the supraorbital and frontal arteries and veins until all the bleeding points are secured. The periosteum is then elevated, most of the separation being confined to the lower flap. If the anterior wall of the frontal sinus is found to be intact it is entered just below the inner extremity of the eyebrow with a mastoid gouge, a motor-driven burr or a mallet and chisel. The opening is enlarged with a Kerrison bone forceps but its extent confined to the minimal requirements of the specific situation.

After attending to the lesions within the frontal sinus, a probe is introduced from the frontal sinus into the ostium in order to locate its position. Antrum rasps then are passed downward from the sinus through the ostium and to the nasal cavity. When the passage has been enlarged to accommodate a tube of 10 mm. diameter, a malleable probe, with an eye in its olive tip, is threaded with heavy silk ligature and passed from the sinus through the enlarged ostium to the nasal floor where the ligature is grasped with a hemostat and the probe withdrawn, thus carrying the two free ends of the ligature to the frontal sinus. A rubber tube, long enough to reach from the lateral margin of the sinus to the floor of the nose is then attached to the nasal end of the ligature and its upper end drawn into the frontal sinus where it is guided in a gentle curve to the lateral extension of the sinus. That portion of the tube lying in the frontal sinus is fenestrated. It is often advantageous to remove the anterior portion of the middle turbinate to facilitate these manipulations. The profuse hemorrhage which occurs during the rasping of the passage subsides promptly as soon as the rubber tube is in position. The lower end of the tube is then cut off so that it terminates in the nasal vestibule. After careful approximation of the periosteal edges, the skin margins are approximated with No. 14 metal clips. The conspicuous ridge produced by the clips prevents separation of the skin margins during postoperative edema and minimizes subsequent scarring. After operation, the frontal sinus can be readily irrigated at any time by passing a No. 8 or 10 French soft rubber catheter through the drainage tube and lavage carried out with normal saline solution or azochloramide solution. The return of the irrigating fluids is thus free and no pressure is produced within the sinus. The drainage tube can be left in place for long intervals without discomfort, but usually epithelialization of the new tract is complete in six weeks or less. Removal of tube is accomplished by grasping the end in the nasal vestibule and pulling it out. No resistance is noted and there is no pain or discomfort during its removal.

A case history is presented to illustrate the results of this simple operative approach to the frontal sinus in the presence of several complicating factors.

REPORT OF A CASE.

A. R., a white woman aged 50 years, was admitted to the University Hospital on Dec. 9, 1949. She had had severe, boring pain in the left frontal region for five days, and there was edema and tenderness over the left frontal region, left cheek and left orbital rims. Admission was precipitated by the appearance of stiffness of the neck suggesting early meningitis.

She stated that she had been relatively well until October, 1948, when she developed severe left frontal pain, followed by swelling and tenderness over the left frontal area and edema of the left upper lid. Following the administration of penicillin and catheterization and irrigation of the left frontal sinus, she became symptom-free. In April, 1949, a similar attack occurred, but she again recovered after further catheterization and irrigation of the sinus and antibiotic therapy. Excruciating pain in the left frontal region again appeared seven weeks prior to the admission to this hospital. For five days the temperature ranged from 101.2° to 104° F. but returned to normal after intensive antibiotic therapy. At this time Roentgenograms revealed the presence of an osteoma in the posterior wall of the left frontal sinus.

On admission the left frontal area was not only swollen and injected, but a doughy character of the soft tissues could be palpated well beyond the hair line. The swollen lids completely closed the left eye, but there were no restrictions on extraocular movements. The left nostril was obstructed, and profuse purulent secretion was present in the left anterior middle meatus. Palpation also detected fluctuation and an apparent depression in the region of the frontal bone, and pus was aspirated with a needle. The spinal fluid was normal; there were 12 per cent neutrophiles and 23 per cent lymphocytes present in the blood with a W. B. C. of 12,600. During the first 24 hours after admission the temperature varied from 100.4° to 102° F., but remained normal thereafter.

At this time Roentgenograms revealed bone absorption in the left frontal region and extending as far posterior as the coronal suture. The report also pointed out that with chemotherapy manifestations of osteomyelitis of the skull have changed so that slight deossification is now the usual finding rather than the true "moth-eaten" appearance described in the past. The right frontal sinus was normal; the left frontal sinus was indistinct in outline with osteomyelitis alterations about its periphery. Dense clouding of the ethmoid cells, particularly on the left, was noted, as well as marked mucous membrane thickening in the right maxillary sinus and slight mucous membrane thickening in the left maxillary sinus. The sphenoidal sinuses were well developed with bilateral mucous membrane thickening.

Four hours after admission, the left frontal sinus was entered and thick, yellow pus escaped under considerable pressure. The anterior wall of the sinus was soft at the site of entrance and the mucosa within the sinus was so edematous as to fill the space. An osteoma 8 x 10 x 2 mm. was located in the posterior wall of the sinus and removed, exposing normal dura beneath. Temporary drainage was secured with the insertion of a rubber tube and the wound closed. The sinus was irrigated with azochloramide solution every six hours; 300,000 units of crystacillin were given intramuscularly every 24 hours and 500 mgm. of aureomycin were given every three hours by mouth. The patient remained afebrile.

Seventeen days after the first operation, the frontal sinus was reentered, the nasofrontal passage enlarged and a rubber tube introduced so as to reach from the lateral margin of the sinus to the nasal vestibule. The external wound was closed with metal clips. Two weeks later, a red, tender, fluctuant swelling appeared over the left frontal area near the hair line. When the pus aspirated from this abscess was cultured no growth was obtained. After further aspiration on two successive days, the swelling disappeared and the pain subsided. At this time the Roentgenograms showed still further destruction in the left frontal bone and a sequestrum appeared to be forming.

The patient remained free of symptoms and the drainage tube was removed at the end of six weeks. During the next three months antibiotic therapy was continued. Three hundred thousand units of crystacillin were administered every 12 hours and 50,000 units of penicillin every three hours intramuscularly, for six weeks after the final operation. During the following six weeks the patient received 500 mgm. of aureomycin by mouth every three hours and 300,000 units of crystacillin intramuscularly daily. All therapy was discontinued thereafter.

By Feb. 7, 1950, the zone of osteomyelitis could no longer be clearly delineated in the Roentgenograms, and on March 3, 1950, no reactivation of the inflammatory process could be observed in the films. Studies on May 5, 1950, revealed the bone defect in the left frontal region to be smaller; however, there continued to be a radiolucent area above the operative site, and although it appeared smaller than at previous examinations it seemed still to contain a small sequestrum which had decreased in size. In November, 1950, further X-ray studies revealed diminished size of the defect in the left frontal region, and the small sequestrum could not be identified. Since Dec. 6, 1950, Roentgenograms have failed to reveal a radiolucent area in the left frontal bone. The patient has been symptom-free for 15 months; the drainage tract from the left frontal sinus has not constricted and no abnormal secretions have drained from it.

The simplest external approach to the frontal sinus is adequate in the management of mucoceles, pyoceles and cysts of the frontal sinus and useful in the removal of foreign bodies and small osteomas. In selected cases it may also suffice in the control of osteomyelitis of the frontal bone.

Successful end-results depend upon permanent, continuous *drainage of the frontal sinus, supplemented with the appropriate* antibiotic agent in adequate dosage and administered over a long period of time or at least until Roentgenologic evidence of infection disappears.

The permanent drainage tract from the frontal sinus to the nasal cavity should be slightly more than 10 mm. in diameter. A rubber tube of 8 to 10 mm. diameter, if left in position within the tract for six weeks, will permit the regenerative ability of the epithelial cells of the nasal and sinus cavities to cover the surfaces of the new channel within a period of six weeks. Practically no deformity results.

LETTER TO THE EDITOR.

C.I.D. AUDITORY TESTS W-1 and W-2.*

Central Institute for the Deaf, with the collaboration of Technisonic Studios, has prepared two new recorded spondaic word tests that resemble the familiar Auditory Tests No. 9 and No. 14 of the Psycho-Acoustic Laboratory (Harvard). These recordings are intended for use with speech audiometers for determining the threshold for speech. The new tests differ from PAL Tests Nos. 9 and 14 in that the vocabulary is confined to very familiar words, suitable for children as well as adults, and also the pace is faster and more suitable for talk-back instead of write-down responses.

The test material in both W-1† and W-2 is a list of 36 spondaic words (like *baseball* and *railroad*) which were selected from the larger group used in Psycho-Acoustic Laboratory Auditory Tests No. 9 and No. 14. The particular words were chosen because they are familiar and because these particular recorded items proved experimentally to be equally intelligible. Six different scramblings of the same list of 36 words have been recorded on discs. The items in each scrambling are identical transcriptions from the same original. The scrambling was done by cutting and splicing the magnetic tape on which the transcriptions of the original were recorded.

In Auditory Test W-1 all of the words are recorded at a constant intensity and they monitor at approximately the same place on a VU meter. Each test word is introduced by the carrier phrase "Say the word." The carrier phrase, and also the 1000 cps calibration tone on the inner band of every record, have been recorded approximately 10 db above the average level of the test words. Auditory Test W-1 is designed for use with a step-attenuator to obtain the greatest possible flexibility and speed in finding thresholds for speech.

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*This work was initiated under Contract N6onr-272 (Project No. NR142-170, T.O. III) with the Office of Naval Research and completed under Contract V1001M-577 with the Veterans Administration.

†Means Word Test Number 1.

Auditory Test W-2 is a "descending level" test. The words in each of the lists are already attenuated on the record. After the first nine words the carrier phrases also start descending but they stay 6 db above the test words. The first group of words starts at the level of the calibration tone. Three words are at this level, the next three are down 3 db, the following three are down 6 db, and so on. Thus an average of 1 db attenuation per test word has been put into each list and each word that a listener repeats correctly lowers his threshold score by 1 db.

A subject's hearing loss for speech is defined as the number of decibels by which his threshold for speech is higher than the experimentally determined average threshold for a number of individuals whose hearing for pure tones in the speech range is within ±5 db of the accepted norms for pure-tone audiometry. The value of the normal threshold for speech will vary not only with the particular sample of speech that is selected as test material but also with the equivalent that is employed. Neither the apparatus for speech audiometry nor the recordings of test material have yet been standardized, either by the Council on Physical Medicine and Rehabilitation of the American Medical Association or by the American Standards Association. It is, therefore, essential for each operator to establish his own normal values for each type of test with his own equipment and under his own particular acoustic conditions; however, if the recommendations concerning apparatus that are given in the manual that accompanies the new recordings are followed, normal threshold values obtained at other clinics should not differ from our own by more than 2 or 3 db. Our tentative values for the normal thresholds of speech are 20 db re 0.0002 dyne per cm^2 for Test W-1 and 19 db re 0.0002 dyne per cm^2 for Test W-2. A more extensive study is in progress to determine whether the small difference between the two figures represents an advantage for the progressive attenuation employed in Test W-2 or whether it represents a difference in the two groups of individuals employed for the two tests.

According to our preliminary measurements a speech audiometer that is calibrated for hearing loss should give a sound level of 20 db re 0.0002 dyne per cm^2 (measured in a National Bureau of Standards 9A coupler) when the calibration tone is set to the reference level on the meter and with the hearing-loss dial at zero. The starting level indicated on the hearing-loss dial minus the number of words of Test W-2 correctly repeated will then give the listener's hearing loss for speech directly within an accuracy of ±2 db. A tone of 1000 cps at 20 db above 0.0002 dyne per cm^2 (under a receiver on the ear or on a 9-A coupler) corresponds approximately to the 1000 cps tone delivered by a properly calibrated pure-tone audiometer at the 5 db hearing-loss setting. This relationship is reasonable because even the most intelligible words must be a few decibels above the threshold of detectability before they can be understood.

The Technisonic Studios made the original recordings of the test material and also have carried through the entire process of manufacture including the electroplating and pressing. The Technisonic Studios will also distribute the recordings, on a non-profit basis, at the following prices:

W-2 as a set. 3 double-faced 78 rpm records, each containing 2 lists of W-1 @	$10.50
W-2 as a set. 3 double-faced 78 rpm records, each containing 2 lists of W-2 @	$10.50
W-1 and W-2 as a set. 3 double-faced 33⅓ rpm records @	$10.50

Individual records from the 33⅓ rpm set may also be purchased, as follows:

1 double-faced 33⅓ rpm record containing 4 lists of W-1 @	$ 4.00
1 double-faced 33⅓ rpm record containing 4 lists of W-2 @	$ 4.00
1 double-faced 33⅓ rpm record containing 2 lists of W-1 on one face, 2 lists of W-2 on the other face @	$ 4.00

All recordings are on 12-inch discs.

The address of the Technisonic Studios is 1201 South Brentwood Boulevard, Richmond Heights 17, Missouri.

We hope that other laboratories and clinics that are interested in the standardization of speech audiometry will try these new recordings and communicate to us their criticisms and suggestions, and also any data that they obtain concerning the normal values for the thresholds of speech.

Signed: R. W. BENSON
H. DAVIS
C. E. HARRISON
I. J. HIRSCH
E. G. REYNOLDS
S. R. SILVERMAN

ILEME CONGRES FRANCAIS D'OTO-RHINO-LARYNGOLOGIE.

The Congrès Francais d'Oto-Rhino-Laryngologie will be held in the Grand Amphithéâtre of the Faculté of Médicine, Paris, Oct. 22, 23, 24 and 25, 1951, under the direction of the presiding Dr. M. Bouchet, of Paris, and Honorary President de M. le Prof. Léon Binet, member of the Institute.

For further information, address Dr. H. Flurin, Secrétary Général — 19 Avenue MacMahon, 17e, or Dr. H. Guillon, Assistant Sécretary Général, 6 Avenue MacMahon, Paris, 17e.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

July 1, 1951.

Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.

Manufacturer: The Dahlberg Co, 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St.,,Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.

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Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.

Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.
Distributor: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.

Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.

Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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NORTH AMERICAN BLASTOMYCOSIS. A REVIEW OF THE LITERATURE AND A REPORT OF TWO CASES PRIMARY IN THE LARYNX.[*]

GEORGE B. FERGUSON, M.D.,
Durham, N. C.

This paper endeavors to point out the clinical and pathological similarities between primary laryngeal blastomycosis and laryngeal cancer. It presents a summary and brief analysis of the general literature concerning systemic blastomycosis, a more detailed investigation of those reports dealing with primary laryngeal blastomycosis, and a report of two cases, in each of which a mistaken diagnosis was made.

DEFINITION.

This discussion is limited to the North American form of blastomycosis (Gilchrist and Stokes, 1898) and avoids consideration of the European form, caused by a fungus of the genus cryptococcus, and of the South American form, which is caused by blastomyces brasiliensis.

HISTORICAL.

Gilchrist and Stokes[1] first described a case of cutaneous blastomycosis in 1894. They named the causative organism

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[*] Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., 1951.

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blastomyces dermatitidis, and were able to demonstrate it in stained tissue sections. Walker and Montgomery[2] described the first case of systemic blastomycosis in 1902. Since then there have been reports of approximately 380 cases, 73 of which have been of the systemic variety.[3] In 1914, Stober[4] and his co-observers in the Chicago area published a monumental work concerning the cases seen there.

In 1918, Dennis[5] made the first report of primary laryngeal blastomycosis. In 1926, Jackson[6] reported a single case with laryngeal and pulmonary infection, and New[7] listed two primary laryngeal cases in 1928. In 1936, in an article titled "Laryngeal Blastomycosis Complicating Carcinoma," Fuller[8] reported a case which, in the light of the conclusions of the present paper, is of particular interest.

OCCURRENCE.

North American blastomycosis is found widely distributed throughout the United States and Canada. For many years the disease seemed centered around Chicago, and was at one time referred to as "Chicago disease," since 50 per cent of all the reported cases resided in that city or in its environs. It has recently been recognized, with increasing frequency, in the southeastern states. Smith[9] reported 59 patients with blastomycosis in 250,000 admissions to the Duke Clinic in 15 years. He explains this unusual frequency of occurrence by stating that "the southeastern states constitute an endemic area for this disease." It seems possible, however, that cases of blastomycosis sometimes occur more frequently in other areas than is recognized. Wade and Bel,[10] for instance, discovered five cases over a period of two years in New Orleans, where none had previously been recognized.

Though no age is immune, 50 per cent of all cases occur in individuals over 40 years of age, and males are affected nine times more frequently than are females.

Dennis[11] and Stober[12] stress the effect of occupation and living conditions on the onset and progress of the disease.

Damp, dusty, moldy rooms and generally poor living conditions seem to predispose to blastomycosis. There is no evidence of transfer by contagion.

MYCOLOGY.

The technique of microscopic examination for fungus with slide preparations of potassium hydroxide and scrapings, pus, sputum, or other suspected material, is fully described in the "Manual of Clinical Mycology,"[13] from which the authors and the W. B. Saunders Co. have permitted me to reproduce three plates illustrating: *1.* blastomycosis dermatitidis as it appears in these microscopic preparations, *2.* the growth obtained on beef glucose agar at 37° C. for 23 days, and *3.* a colony on Sabouraud's glucose agar at room temperature for 23 days (see Figs. 1, 2 and 3).

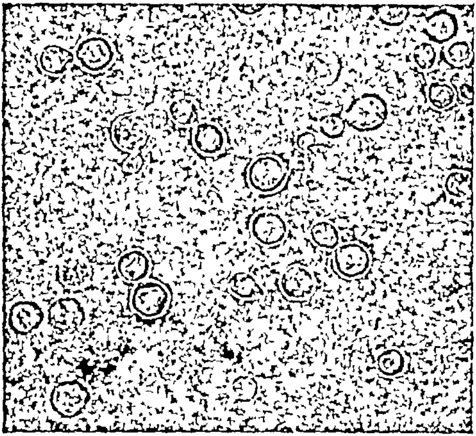

Fig. 1. Round, double-contoured, budding yeast-like cells in pus from subcutaneous abscess. × 700.

PATHOLOGY.

Pathology textbooks devote little space to blastomycosis, and some fail even to mention epithelial hypertrophy as a prominent feature. This last omission is perhaps accounted for by the fact that much of their material for study is obtained at postmortem examinations, or from generous biopsy specimens which permit a study of the disease process of the organ as a whole. Eggston and Wolf,[14], writing a specialized textbook of ear, nose and throat pathology, point out the possibility of

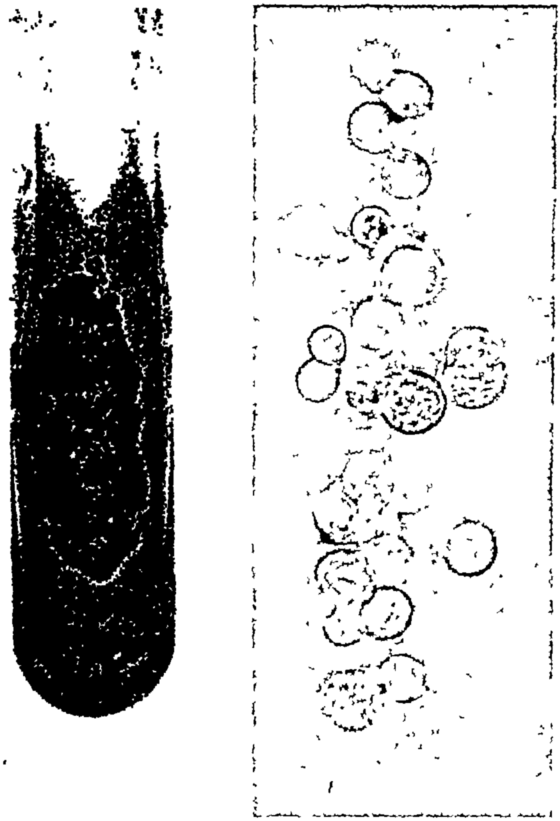

Fig. 2 (a) Yeast-like culture, 21 days, on beef infusion glucose agar at 37° C. (b) Budding yeast-like cells from beef infusion glucose agar at 37° C. × 700

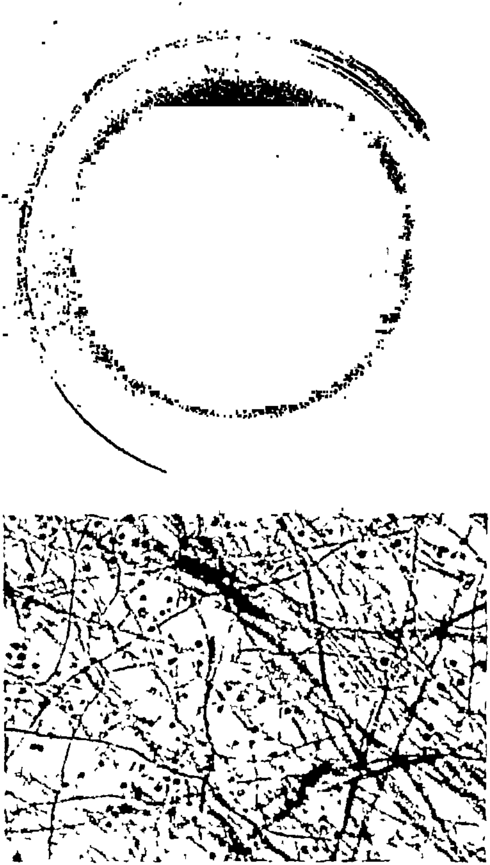

Fig. 3 (a) Filamentous colony on Sabouraud's glucose agar, 23 days, at room temperature (b) Round and pyriform conidia found in filamentous stage from Sabouraud's glucose agar at room temperature. × 700.

confusion with carcinoma. Constant experience with the difficulties inherent in the interpretation of specimens which are small and often far from all inclusive, such as those obtained from biopsy of the larynx, may add to one's awareness of the dangers of diagnostic misinterpretation. There is, of course, great variance in the extent of the epithelial hypertrophy itself.

Undoubtedly the tissue reaction to injury by blastomyces dermatitidis varies widely, depending upon the host's immunologic reactions and the sites of invasion. The most characteristic response, as noted by most observers, is multiple small abscess formation. Giant cells are common, and the organism may be found within them; in some areas the organism cannot be demonstrated. Caseous necrosis, tubercle formation, and fibrosis may be prominent. The discovery of spherical, double-contoured, budding, thick-walled organisms eight to 15 micra in diameter is considered by some to be diagnostic.

IMMUNOLOGY.

Hypersensitivity to the antigen of blastomyces may be demonstrated by a positive skin test to blastomyces vaccine, and humoral antibodies can be detected by a complement fixation test. D. T. Smith,[15] who feels that the prognosis in blastomycosis depends on the immunologic state, studied 40 cases of blastomycosis at the Duke Clinic, and found the following immunologic groups:

1. *Skin test positive—complement fixation negative.* Prognosis in this group is relatively good, since these patients respond well to treatment after desensitization with vaccine.
2. *Skin test positive—complement fixation positive.* The response to treatment in this group is not so satisfactory, but may be good following desensitization.
3. *Skin test negative — complement fixation positive.* The prognosis in this group is very poor, as this represents an anergic reaction.

4. *Skin test negative—complement fixation negative.* These are usually early cases in which hypersensitivity and complement-fixing antibodies have not yet developed. Obviously, this group of patients will ultimately reclassify themselves into one of the other three groups.

DIFFERENTIAL DIAGNOSIS.

The usual portal of entry of systemic blastomycosis is the lungs—Wade and Bel[16] state that 96 per cent of all autopsied cases having blastomycosis had lung involvement—and the lesions greatly resemble tuberculosis. The onset is usually ascribed to "taking a cold." Dyspnea, pain in the chest, cough, and expectoration of purulent or bloody sputum are frequent complaints. The duration may be from a few months to several years. Anyone suspecting blastomycosis would, therefore, expect to find lung involvement; however, such is not always the case, and one must be on the alert to recognize the disease when no lung involvement occurs.

The similarity to tuberculosis is noted even in localized sections, such as those involving the larynx. Other diseases to be differentiated are lues, Boeck's sarcoid, other fungus infections, and, as found in my own two cases, cancer. In these two cases, as will be subsequently noted, both the clinical and pathological picture greatly resembled carcinoma.

PROGNOSIS.

All observers have noted a marked difference between the two main forms of blastomycosis, so far as the prognosis is concerned. The common cutaneous type rarely causes death, and rarely spreads to become the systemic variety. The systemic variety is usually fatal. Martin and Smith[17] noted a 92 per cent mortality rate in such cases when they had been followed two years or longer.

TREATMENT.

It is obvious from the high mortality rate in systemic blastomycosis that the treatment of this form of the disease is confusion with carcinoma. Constant experience with the difficulties inherent in the interpretation of specimens which are small and often far from all inclusive, such as those obtained from biopsy of the larynx, may add to one's awareness of the dangers of diagnostic misinterpretation. There is, of course, great variance in the extent of the epithelial hypertrophy itself.

Undoubtedly the tissue reaction to injury by blastomyces dermatitidis varies widely, depending upon the host's immunologic reactions and the sites of invasion. The most characteristic response, as noted by most observers, is multiple small abscess formation. Giant cells are common, and the organism may be found within them; in some areas the organism cannot be demonstrated. Caseous necrosis, tubercle formation, and fibrosis may be prominent. The discovery of spherical, double-contoured, budding, thick-walled organisms eight to 15 micra in diameter is considered by some to be diagnostic.

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Case 2: In 1923, Dr. Chevalier Jackson reported the case of a 48 year-old white male who worked as a clerk in a general store Hoarseness, which began two years previously, became progressively worse, and dyspnea developed. The patient was told he had pulmonary and laryngeal tuberculosis and asthma. He was unrelieved by a change in climate and, the dyspnea becoming worse in spite of epinephrine injections, a tracheotomy was performed. At another institution the patient's illness was diagnosed as malignancy, but the pathologist's report failed to confirm this diagnosis.

At the time Dr Jackson saw and described the case there was a purplish red nodular area of inflammation of the skin about the tracheotomy wound The patient complained of burning and soreness of the skin and noted slight cough and expectoration of a small amount of mucopurulent secretion He had no fever, and had had slight weight loss.

The following is a digest of Dr. Jackson's description of the mirror appearance of the larynx· An infiltrative lesion involved the left side of the larynx, part of the right side, and the interarytenoid space The left cord and the ventricular bands were not distinguishable because of the presence of a grayish, granular mass, which looked as though it had been brushed with silver nitrate. The left arytenoid was fixed, and the arytenoid half of the epiglottic fold was swollen. The right cord was replaced by a nodular mass which extended from the anterior to the posterior commissure, and was described as having a glistening "frog spawn" appearance

Biopsy from the larynx showed inflamed and ulcerated mucosa, with considerable epithelial hyperplasia. Fibrosis, and numerous small foci containing giant cells, were present in the submucous tissue, giving "somewhat the appearance of a tuberculous lesion." Biopsy of the skin about the tracheotomy wound revealed blastomycosis The patient was treated with potassium iodide

Case 3· Dr Gordon B New reported two cases in 1928* The first, seen in 1919, was a 51-year-old farmer who, at the time of examination, had been hoarse for 18 months. In 1902, following an attack of influenza, he had been hoarse for six months, and slight shortness of breath had persisted to the present illness. The patient complained of some cough, and aphonia had developed a few days prior to Dr New's examination. There had been a weight loss of 35 pounds in the last two years. Chest X-ray, Wassermann, and sputum examinations were all negative

The larynx was described as showing some scarring on both vocal cords The glottis was small, and a small web was noted at the left vocal angle. There was a roughened area in the interarytenoid area A small, grayish nodule was present on the right vocal cord near the middle, and both cords were somewhat inflamed and thickened

Biopsy revealed inflammatory tissue only, with marked epithelial fibrosis Tracheotomy was done because of dyspnea. Dermatitis developed around the tracheal opening, and a biopsy from the skin showed blastomycosis. Later biopsy of the larynx also showed blastomycosis.

The patient gained weight and improved rapidly in voice and general health on potassium iodide and X-ray therapy. The larynx remained somewhat scarred and thickened, but no evidence of activity of the blastomycotic lesion was noted at subsequent examinations. He was apparently well in 1928.

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* Dr New reviewed an additional case of primary fungus infection reported by Sartory[11] this however, is of the European cryptococcus type (and I do not include it)

Case 4: Dr. New's second case was a 35-year-old iceman who, at the time of his examination on Nov. 16, 1927, had been hoarse for seven months. He described the onset as an acute cold; hoarseness and dysphagia gradually developed. The patient had had no night sweats, cough, or fever. Tuberculosis had been diagnosed by previous physicians, and repeated biopsies elsewhere had revealed only inflammatory tissue.

The chest X-ray, Wassermann and sputum examinations were negative. The larynx was described as showing an extensive chronic process involving both sides. The right false cord and the anterior part of the left were described as "mammillated in appearance," and some areas were covered by a thin grayish membrane. Both arytenoids were somewhat swollen and restricted in motion.

The biopsy, taken by Dr. New, was reported to show changes resembling tuberculosis; however, on further study, a diagnosis of blastomycosis was made.

The patient took massive doses of potassium iodide and showed marked improvement.

Case 5: In 1936, Dr. T. E. Fuller reported the case of a 71-year-old male who came under his observation on March 7, 1930. The patient dated the onset of throat discomfort to an attack of influenza suffered in January of that year. Examination on March 7 was essentially negative, except for the vocal cords. They were described as "dull," and "the picture was exactly what we see in people who have smoked to excess for a long time."

The patient was next seen in September. He was hoarse and had great difficulty in swallowing. Dr. Fuller states that "examination revealed a large cauliflower mass involving the right arytenoid, the interarytenoid space, the aryepiglottic fold, the lower part of the epiglottis, and the base of the tongue." No enlarged lymphatic glands were present. The Wassermann was negative, and no evidence of tuberculosis was noted. Dr. Fuller's clinical impression was carcinoma. A consultant agreed but felt that, because of the patient's age, irradiation with radium, rather than surgery, should be tried. This treatment was refused, and shortly gastrostomy, and later tracheotomy, was required.

By the Spring of 1931 there was swelling of the glands in the right side of the neck attributed to carcinoma; however, as fluctuation occurred, the area was incised, and pus was obtained. Several draining sinuses formed, around which there was extensive granulation. Sections were taken from the granulating areas and reported as follows: "Section shows hyperplasia of squamous cell epithelium with irregular papillary arrangement and rather incomplete differentiation, in that hyalinization is not prominent. Cells vary somewhat in size, shape, and staining qualities, and show occasional mitotic figures. The basement membrane, however, is in general sharp. The stroma is edematous and throughout infiltrated with leucocytes, lymphocytes and endothelial cells. . . ."

Cultures from the pus gave a growth of blastomyces dermatitidis.

The patient died on July 15, 1932, and a partial autopsy was done. A large retropharyngeal abscess was located, which probably extended into the mediastinum. The pathologist reported that he could find only chronic inflammatory tissue. At a later date more sections were removed from the gross specimen, however, and the pathologist reported squamous cell carcinoma Grade IV.

Dr. Fuller reports this case as a "low grade of carcinoma which probably began in the postcricoid region and that the infection by the blastomycetes was secondary, producing the deep abscess and the toxemia from which he died. On the other hand, it is possible that the blastomycosis may have been primary, and that the carcinoma resulted from the chronic inflammation."

COMMENT.

Difficulty has been encountered in making a diagnosis of blastomycosis from laryngeal biopsy.

In Case 1, alone of this group, the patient gives a history of probable direct exposure to blastomycosis—*i.e.*, the rotting, decaying material of the grain elevator. Although the patient was first clinically diagnosed as having tuberculosis, in this case the laryngeal biopsy was immediately diagnosed as blastomycosis.

In Case 2, and in Case 3, in both of which the laryngeal biopsies had been inconclusive, the biopsy diagnosed as blastomycosis was obtained from skin lesions surrounding the tracheotomy wound. In Case 3, a later laryngeal biopsy, taken after that of the skin lesion, was also considered to be positive for blastomycosis.

In Case 4, as given by Dr. New, the biopsy was diagnosed as blastomycosis only after considerable study, while repeated biopsies elsewhere revealed only inflammatory tissue.

It would be extremely interesting to re-examine the slides of Case 5. While it is possible that blastomycosis and Grade IV carcinoma coexisted, the blastomycosis was undoubtedly present, and almost undoubtedly primary. Since blastomycosis may produce epithelial hyperplasia which may be confused with cancer, it seems at least possible, in the light of later cases, that blastomycosis itself was the sole lesion.

In three of these five cases the authors' descriptions of the clinical appearance of the larynx greatly resemble the description considered typical for cancer.

Case 2 is described by Dr. Jackson in such terms as "infiltrative lesion," "nodular mass" and "fixed arytenoid," all terms clinically descriptive of cancer. Indeed, malignancy had been the clinical diagnosis of another institution.

Case 3 is described as having "a small grayish nodule present on the rignt vocal cord" and the vocal cords are described as being "inflamed and thickened."

The lesion in Case 5 is described as "a cauliflower mass" and Dr. Fuller's diagnosis was that of carcinoma.

The clinical description of Case 1 and of Case 4 most closely resembles tuberculosis.

Both Dr. New and Dr. Jackson noted a grayish appearance in the blastomycotic lesions. Dr. Jackson described the lesion as "looking as though brushed with silver nitrate." This may possibly be a helpful differentiation feature in the diagnosis of blastomycosis.

REPORT OF TWO CASES IN WHICH MISTAKEN DIAGNOSIS WAS MADE.

Case 1: On Nov. 24, 1947, a letter was received referring a 58-year-old white male cabinet maker as a service patient to the ear, nose, and throat clinic of the Duke Hospital. The patient, however, failed to present himself until Dec. 2, 1947, at which time he was examined in the outpatient dispensary by a member of the resident staff. The referring physician had made two previous biopsies of the patient's larynx. He sent us the pathologist's report of the second biopsy, which was diagnosed as squamous cell carcinoma.

At the first visit, the patient gave a history of having been perfectly well until two months before, at which time he had developed bronchitis and laryngitis accompanied by a tickling sensation in the throat, and a marked desire to cough. He had been unable to speak above a whisper for the past six weeks and recently had noticed slight dysphagia. Mirror examination was noted as showing a normal epiglottis. There was some restriction of motion of the right vocal cord, and slight edema of both arytenoids and ventricular bands. The anterior one-half of each vocal cord was obscured by the swelling, and the remaining portions were noted to be ragged in appearance. A few "shotty," freely movable cervical lymphatic glands were palpated in the neck. The remainder of the nose and throat examination was negative, except for dental infection. Kahn, Kline, and Mazzini tests taken at this examination were reported negative.

The patient was given an appointment for hospital admission, but failed to return on the specified date. Finally, on Dec. 17, 1947, his admission was completed Physical examination, including chest X rays, proved negative, and on Dec. 18, 1947, in my absence, after consultation with another staff physician, the resident physician performed a total laryngectomy

I saw the patient during his convalescence, which was uneventful. Primary healing of the wound was obtained, and the patient was discharged on Dec 27, 1947, the ninth postoperative day The patient's temperature on the evening of admission was recorded as 100.2° F On the morning of operation it was 97.8° F., and rose to a maximum of 101° following operation. It returned to normal on the fifth postoperative day Penicillin in a dosage of 20,000 units was given every three hours for the first six days

At the time of operation the larynx was submitted to the pathologists at Duke Hospital The following quotations are from their report on gross and microscopic pathology "The specimen consists of a larynx, the mucosal covering of the larynx is involved in a leucoplakic lesion which has irregular surfaces The vocal cords cannot be distinguished due to edema and to tumor involvement. All sections show essentially the same picture The epithelium shows some down growth in places but there is a normal maturation pattern as one progresses from the basal layer to the surface This lesion is interpreted as a hyperplasia of the epithelium Within the epithelium in some places there may be seen small abscesses, and occasionally one may see giant cells In many of the giant cells . there are refractile, double-contoured bodies" (see Fig. 4)

The previous biopsy slide was obtained from the original pathologist and was reviewed All sections showed the same lesion, and on Feb 15, 1948, the diagnosis was altered from cancer to blastomycosis (see Fig 5)

The patient was promptly readmitted on Feb 19, 1948 He had had an uncomplicated course, except for the appearance of a red, indurated skin lesion the size of a 25 cent piece, located on the left side of the face in front of, and below, the ear (see Fig 6) Consultation was obtained with the dermatologist, the immunologist, and the Roentgenologist Scrapings from the skin lesion revealed blastomycetes A biopsy of the skin lesion was diagnosed as chronic dermatitis, since neither blastomycetes nor other characteristic changes could be identified Both skin test and complement fixation test were negative

The patient was started on standard courses of killed blastomyces vaccine 1 1,000 Each standard course consists of the following injections measured in cubic centimeters 0 1, 0.2, 0 4, 0 6, 0 8, 1 0, given twice weekly. The skin lesion increased in size, and the standard course of vaccine was repeated On May 6, 1918, the skin test and the complement test were again negative, and a standard course of 1·100 vaccine was started Since that time the patient has been given numerous standard series of vaccine and, since May 26, he has been under almost constant treatment with potassium iodide X-ray therapy was first given over the lesion near the left ear, and was continued as other areas of skin involvement developed In spite of treatment, the skin lesion has spread (see Fig. 7), however, the complement fixation test, the skin test, and the chest X-ray remain negative

Case 2 A 49 year-old white male druggist, referred by Dr Alan Davidson was first seen by me on Sept. 7, 1918 The patient's only complaint was of hoarseness, which had been present since February, 1948. He gave a family history which was negative for tuberculosis, cancer, and diabetes. He was a man of moderate habits, and smoked less than 20 cigarets daily. His only illnesses had been those usual in childhood.

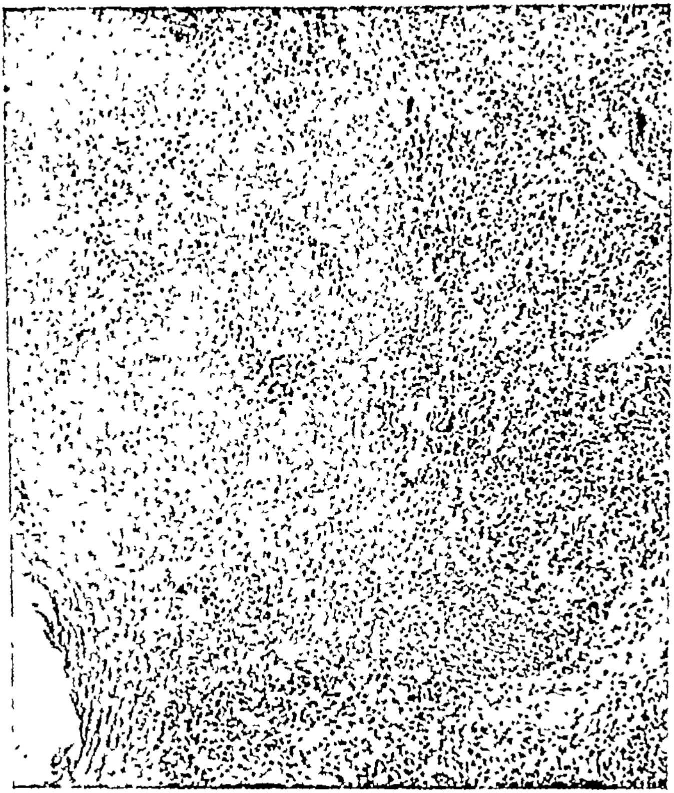

Fig. 4. Case 1. Moderate epithelial hyperplasia.

The patient dated the onset of his present illness to two severe colds; the first of these cleared completely, but the second left him permanently hoarse. The following description of the earlier appearance of the larynx was obtained from the referring physician. When first seen by him five months after the onset of the hoarseness, there was, according to his statement, "a papilloma of the anterior third of the vocal cord, as well as a mass on the anterior surface of the trachea just below the vocal cord. This mass had a whitened, ulcerated area on top of it, and it appeared clinically to be malignant; however, a biopsy taken directly from it was reported back as papilloma."*

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*This biopsy, and a second specimen obtained by the referring physician in July, 1948, were both re-examined by the original pathologist, and no evidence of blastomycosis could be found, his diagnosis remained squamous papilloma

The above quotation is taken from a letter dated Aug 12, 1948 At the time of my examination of the patient on Sept. 7, 1948, he appeared comfortable, cooperative, and was ambulatory He was a moderately spare, middle-aged white male, who had a hoarse voice. Other than throat lesions, no physical abnormality was noted His temperature was 98 4° Laboratory data was as follows Chest X ray, negative, serologic reactions, negative, hemoglobin 85 per cent, R B C, 4,110,000, W B C, 8 800 Mirror examination of the larynx showed the epiglottis to be normal in appearance The arytenoids were not swollen, but the left was somewhat restricted in motion Both vocal cords were diffusely affected by an irregular hyperplasia and appeared reddened, thickened, and irregular The left cord was definitely more involved than the right. No lesion was noted in the trachea at this examination Palpation of the neck revealed one small soft lymph node over the area of the crico thyroid membrane

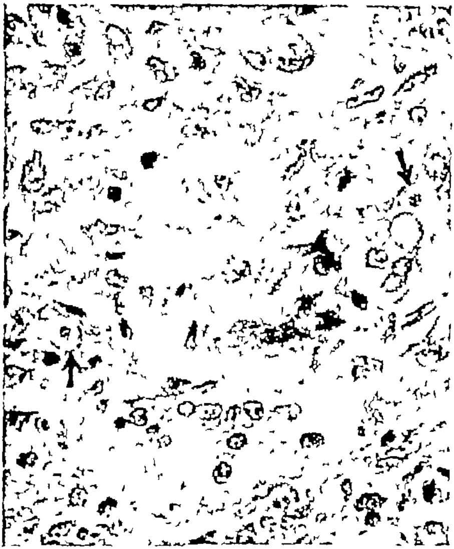

Fig 5 Case 1 Double-contoured, refractile blastomycetes

The patient was admitted to McPherson Hospital on Sept 7, 1948, and direct laryngoscopic examination was performed on the following day

Careful examination of the larynx at that time confirmed the mirror findings. The mucosa below the level of the vocal cords appeared smooth, and I was unable to demonstrate any tracheal lesion. A biopsy specimen was obtained from the middle of the left cord and was submitted to our regular pathologist for microscopic study.

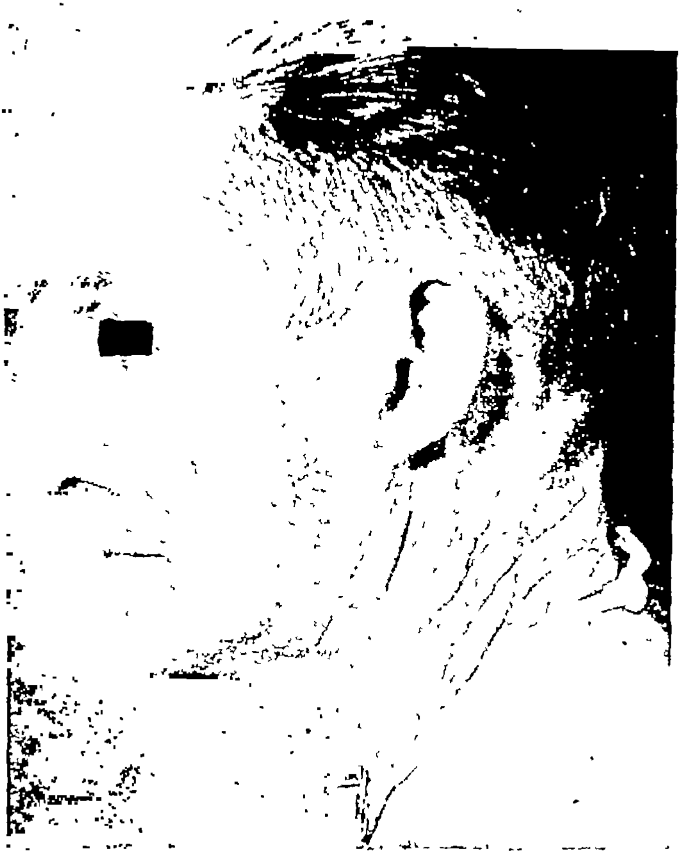

Fig. 6. Case 1 Early blastomycosis of skin.

My impression following this examination was that the lesion was not a benign papilloma. It appeared to be diffuse, invasive, and hyperplastic—probably carcinoma. Since the biopsy taken at the time of examination was diagnosed as cancer, we felt that total laryngectomy was indicated (see Fig. 8).

The patient was discharged from the hospital for a brief interval, and was readmitted on Sept. 12, 1948. Total laryngectomy was performed on Sept. 13. The soft lymph node previously felt at the cricothyroid membrane level together with a wedge of tissue from the thyroid isthmus was excised. The hyoid bone was divided in the midline. The trachea was incised between the first ring and the cricoid cartilage, and the larynx was dissected from below, upwards. The larynx, including the epiglottis,

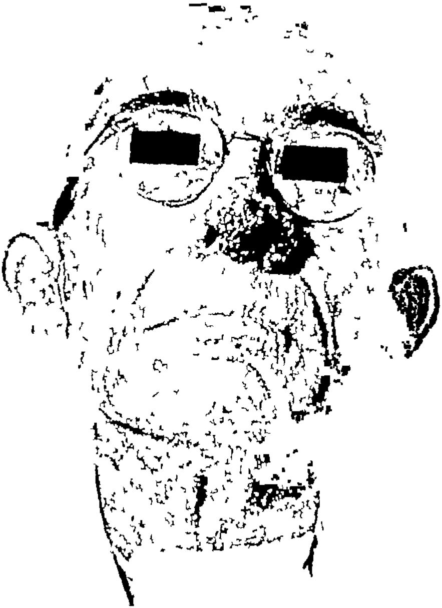

Fig. 7. Case 1. Showing spread of skin blastomycosis.

was excised, together with the pre-epiglottic tissues. These structures were inspected, and seemed to include all the growth; however, when the tracheal stump was examined, the mucosa seemed thickened, with a surface which was faintly granular and somewhat reddened. Because of these abnormalities, I took a specimen of the tracheal mucosa for examination (see Fig. 9).

The usual double row closure of the pharynx was carried out, and the constrictor and ribbon muscles were brought together to reinforce the closure.

The patient's postoperative course was entirely uneventful. Primary healing was obtained, and the patient left the hospital on the eleventh postoperative day. The highest temperature elevation of 100.6° F. occurred on the second day, and the temperature was normal from the sixth day onward. The patient was given a daily injection of 300,000 units of procaine penicillin during his stay in the hospital. On the third postoperative day the pathologist reported the lesion to be blastomycosis rather than cancer, and we added 1 gm. of streptomycin to the daily treatment.

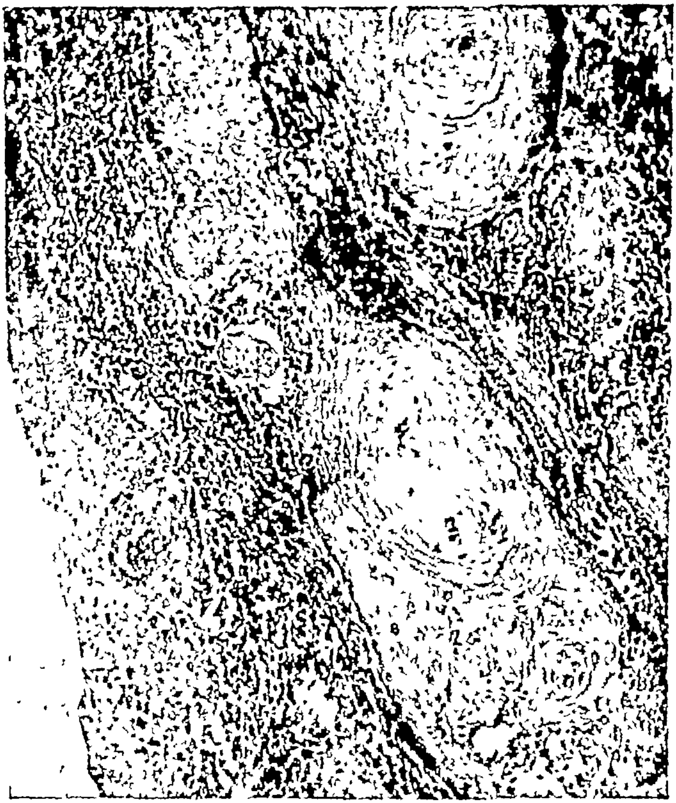

Fig. 8. Case 2. Marked epithelial hyperplasia suggesting cancer.

After discharge from the hospital the patient was immediately referred to Dr. D. T. Smith, Dr. Robert Reeves, and Dr. N. F. Conant for treatment of blastomycosis, and is still under active treatment. Immediately after his discharge his immunologic reactions were determined The skin and the complement fixation test were both negative On Oct. 9, 1948, the patient started a standard course of blastomyces vaccine injections, which were given him by his family physician. He returned for further tests on Nov. 12, 1948 He had gained weight, and the local appearance of the tracheotomy and of the pharynx was normal The standard series of vaccine was repeated By Jan. 1, 1949, the patient noted bloody cough, and examination revealed extensive granulation of the upper trachea, with narrowing of its lumen. Potassium iodide was added to the treatment, and three X-ray treatments of 100 r each were given over the tracheal opening. Marked improvement was noted.

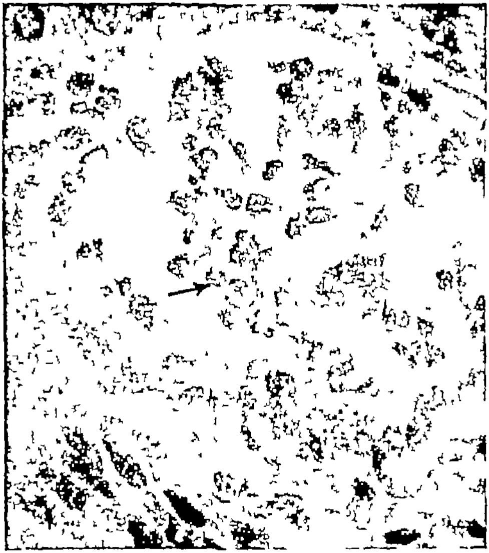

Fig 9 Case 2. Budding blastomycete.

At the present time, slightly more than two years after operation, this man seems to be in relatively good health, he is working, and has maintained his weight. He has developed an excellent pharyngeal voice, his main complaint is of occasional coughing of bloody mucus from the trachea. Chest X-rays have been repeatedly negative He is at present taking blastomyces vaccine and potassium iodide Unfortunately, he has not been able to tolerate large doses of potassium iodide, and frequently has to discontinue it. His last complement fixation test, taken Aug. 9, 1950, was positive in a dilution of 1:4, while the skin test remained negative. This may possibly signify his immunologic reclassification in the unfavorable anergic group.

SUMMARY.

Both the cutaneous and systemic forms of blastomycosis, though rare, are found widely distributed in the U. S. A. and Canada. This paper deals more fully with the less common and far more serious systemic variety, which as shown by the literature, is often mistaken for tuberculosis. Errors in diagnosis may make it seem more rare than is the actual case, for the recognition of one case in a given area sometimes seems to lead to the discovery of others.

All forms of the disease are caused by a fungus, the blastomyces dermatitidis. In tissue, this organism reproduces only by budding. When incubated at 37° C. it produces a yeast-like colony composed of budding cells. At room temperature, a filamentous colony and round and pyriform conidia may be found.

The pathologic changes noted in tissues affected by blastomycosis vary greatly. Giant cells, sometimes containing double-contoured refractile, budding blastomycetes, caseous necrosis and epithelial hypertrophy may vary in extent so that confusion in diagnosis often exists.

A skin test with blastomyces vaccine, and a specific complement fixation test aid in determining the patient's immunologic state and are a valuable guide in treatment and in prognosis. Blastomycosis is resistant to all recent antibiotic and chemotherapeutic agents and responds best to potassium iodide, X-ray, and possibly surgery in certain selected instances.

The similarities between systemic North American blastomycosis and tuberculosis have been frequently pointed out in the literature, while the close similarity of the primary laryngeal form to laryngeal cancer has not. Well-trained pathologists of various institutions have experienced difficulty in arriving at a positive diagnosis of this disease.

CONCLUSIONS.

1. Primary laryngeal blastomycosis may present laryngeal changes clinically indistinguishable from laryngeal cancer.

2. Small biopsy specimens, such as those obtained from the larynx, rarely permit a positive diagnosis of blastomycosis and the epithelial hypertrophy, when marked, may lead to an erroneous diagnosis of cancer.

3. The two patients subjected to laryngectomy are still alive three and two years, respectively, after operation; however, they still show evidence of blastomycosis and, in my opinion, surgical removal of seemingly localized infection should be undertaken only after a thorough study and an ample trial of the usual vaccine, potassium iodide and X-ray treatments has failed to produce cure or marked improvement.

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THE SURGICAL MANAGEMENT OF CARCINOMA OF THE PARANASAL SINUSES.*

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At the present time the most effective treatment for carcinoma of the paranasal sinuses consists in utilizing a combination of radiation and surgery. There is some disagreement, however, as to how these agents should be used together.

Some have advocated the initial use of cancer-lethal doses of X-ray or radium followed by a surgical procedure to provide drainage for the sloughing tumor mass. Windeyer,[1] for example, gives deep X-ray therapy followed in four weeks by surgical removal of the palate and alveolus. Some of his patients are treated further by intracavitary radium. Tod[2] advocates the implantation of a radium tube into the center of the neoplastic mass and uses surgical drainage when necessary. Prolonged cures resulting from deep X-ray therapy alone have been reported by Baclesse,[3] del Regato,[4] Lenz[5] and others. Radiation therapy is not without its complications. When given in effective dosage there is necrosis of soft tissue and often of bone. Surgical drainage is then required to prevent serious sepsis. Bronchopneumonia and meningitis which were serious complications in the past can now be controlled with antibiotics. The threat of ocular complications is great when the ethmoid cells have to be irradiated.

There are also those who prefer an initial surgical procedure followed by radiation. Ohngren[6] and New[7] have used surgical diathermy to destroy the maxilla and its contents. This is followed by deep X-ray or intracavitary radium in most cases. Hautant *et al.*[8] and Collins and Poole[9] have used

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moderately extensive surgical resection of the maxilla and followed with intracavitary radium. Schall[10] and others perform a wide radical removal of the maxilla and adjacent structures and follow with radiation. Wille[11] reported on 220 patients treated at the Norwegian Radium Hospital and concluded that the best therapeutic regime for carcinoma of the maxilla was surgical removal followed immediately by radiation therapy.

It is known that cancer-lethal radiation therapy is followed frequently by sepsis and sequestration and that wide surgical exposure and drainage are necessary. Thus, because eventually some form of surgery is required following radiation therapy, it seems logical that we should resort to surgery in the beginning and that this surgical procedure should be a vigorous attempt to remove the neoplasm completely. If this is followed by cancercidal doses of radiation we have done the most for our patient.

It is realized that a rigid therapeutic regime cannot be applied to all patients. In the first place, 5 to 10 per cent of all paranasal sinus neoplasms are sufficiently radiosensitive to justify the primary use of radiation. Lymphosarcoma and transitional cell carcinoma fall into this group. Margaret Tod,[9] of the Holt Radium Institute, reports 50 per cent five-year survivals of 18 cases of lymphosarcoma treated by X-ray therapy alone. Primary surgical resection should not be attempted when there is no reasonable chance for complete removal of the neoplasm. The criteria for operability of a particular lesion are determined to some extent by the surgeon's skill and courage. There are several contraindications to the definitive surgical removal of a primary malignancy of the paranasal sinuses. *1.* Complete resection is impossible when there is invasion of the base of the skull. *2.* If the neoplasm is adherent to the walls of the nasopharynx it probably cannot be resected. *3.* Surgery is to be avoided if there is extensive bilateral involvement because of the hopeless facial defect which results. *4.* Distant visceral or skeletal metastases almost certainly preclude a cure and complete resection of the primary should not be attempted. 5. The same holds true when there are insurmountable regional lymphatic metastases. In our experience about 30 per cent of all patients with carcinoma of the paranasal sinuses fall into an inoperable group. Such patients should receive palliative radiation therapy. Palliation for two or three years is not unusual and five-year survivals have been reported. After excluding those patients with advanced lesions and those with radiosensitive tumors, there remains a group (comprising 60 to 70 per cent of the total) having squamous cell carcinoma or adenocarcinoma in which excision is possible.

A biopsy must be taken to establish the histological type before treatment is begun. It must be deep enough to avoid necrotic surface tissue. Bleeding may be active but is readily stopped with gelfoam and pressure packing. A needle biopsy taken through the face is to be avoided because of the possibility of creating a suppurating sinus tract and the danger of implanting tumor cells into the facial tissues.

Benign tumors need not be treated in the manner to be described here for malignancy. The mixed tumor and adamantinoma, for example, are inherently benign. It is well known that they recur after incomplete excision and then may be locally destructive and invasive. Although these tumors must be completely removed, the palatodental plate, zygoma, orbital contents and nasal bones often can be preserved. A simple lateral rhinotomy incision (without splitting the lip) or a wide Caldwell-Luc incision usually is adequate for removal of these tumors. The surgical procedure should always provide for a permanent large infralabial fenestra. A soon as the packing has been removed (three to five days) the patient should be provided with a prosthetic appliance for the fenestra as seen in Fig. 1. Such a fenestra permits thorough postoperative examination and makes possible the early detection and treatment of recurrences.

All patients with suspected malignancy of the paranasal sinuses should have Roentgen studies of the skull, including an axial view. In the search for metastases there should be a routine preoperative chest Roentgenogram. Patients who have had nasal hemorrhages may require a preoperative blood transfusion. Depleted plasma proteins can be corrected by administering whole blood or plasma transfusions. Because most carcinomas of the nose and paranasal sinuses are infected, it is important to administer antibiotics preoperatively for four or five days in dosages sufficient to insure an adequate blood level.

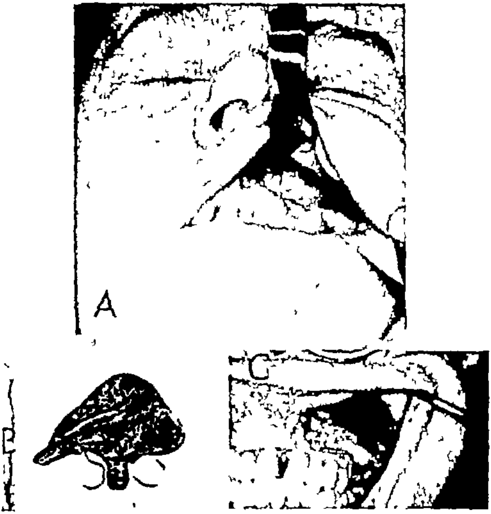

Fig 1. (Mrs. H) Removal of maxilla but with preservation of the palatodental plate (for adamantinoma) (a) View showing the infralabial fenestra. (b) The prosthetic appliance (c) The prosthetic appliance in place

Whether the surgeon uses diathermy or cold steel to perform the resection is not a critical factor. It is important by either method to achieve a wide surgical exposure and to perform a complete resection of the area involved.

If the external carotid artery on the same side as the lesion is ligated first, there is less bleeding during the operation, thus giving a clearer operative field. One to three units of whole blood are usually required during surgery to replace blood loss.

Intravenous pentothal plus intratracheal nitrous oxide-oxygen provides satisfactory anesthesia. A long gauze strip is placed in the pharynx around the intratracheal tube to prevent aspiration of blood and pharyngeal secretions.

BLOCK RESECTION OF THE MAXILLA.

Block resection of the maxilla may be performed in the following manner:

The incision begins midway between the inner canthus and the bridge of the nose as seen in Fig. 2a. It follows the lateral border of the nose, circumvents the nares and extends through the entire thickness of the upper lip in the median or paramedian line. The superior labial and lateral nasal branches of the facial artery must be tied or coagulated during this procedure. An incision is made through the mucosa deep in the gingivolabial fold from the midline to the maxillary tuberosity. A facial flap is elevated so as to leave periosteum, fat and connective tissue on the anterior surface of the maxilla. The lateral wall of the nose is separated from its attachment to the margins of the pyriform aperture, thus exposing the interior of the nose (see Fig. 2, b and c).

The alveolus and bony palate must be removed with the resected block. The mucoperiosteum of the roof of the mouth may be preserved if it has not been invaded by cancer. This membranous flap can be separated from the bone with a heavy mastoid elevator, beginning on the buccal surface of the alveolus (see Fig. 3a). It is necessary to proceed with caution because the periosteum is firmly adherent and perforation can occur easily near the midline where the mucoperiosteal layer is thin. When teeth are present the palatal flap may be elevated from an incision made on the lingual surface of the alveolus (see Fig. 3c). If the neoplasm has involved the palate, the entire thickness must be removed well beyond visible tumor so as to insure complete excision (see Fig. 3b).

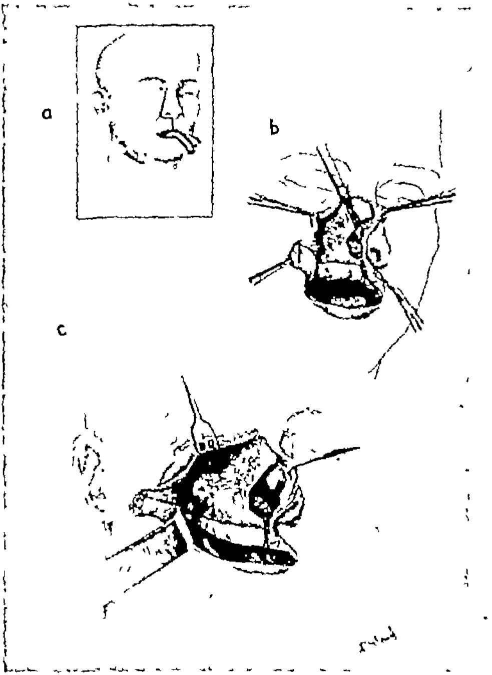

Fig 2 Block resection of the maxilla. (a) Incision (b and c) Exposing the anterior surface of the maxilla and the interior of the nose.

The nasal bone and the frontal process of the maxilla should be removed in most cases. This removal gives wide access to the ethmoidal labyrinth, nose and frontal sinus. If it has been established beyond doubt that the malignancy has involved only the lower part of the maxilla, the frontal process of the maxilla may be divided in its midportion, thus preserving the nasel bones (see Fig. 3, d, e, f).

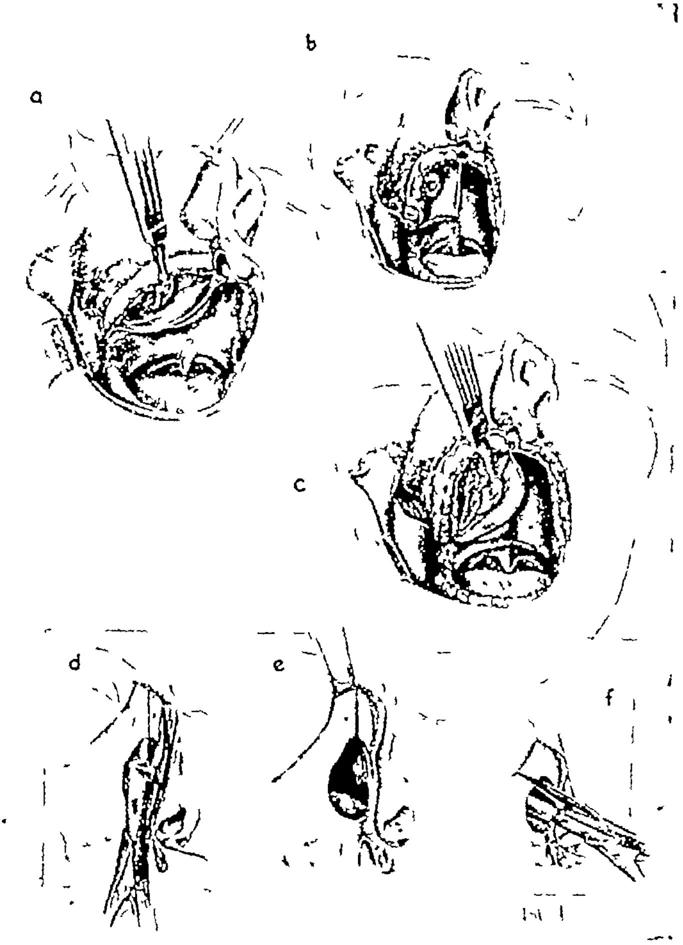

Fig 3 Methods for handling the palate and nasal bone (a) Elevation of a mucoperiosteal flap from the alveolus and hard palate when the palate has not been invaded by cancer (b) Hemisection of the palate when these tissues are involved by neoplasm (c) Elevation of the flap when teeth are present. (d) Separation of the nasal bones in the midline (e) Cutting through the nasal bone and the frontal process of the maxilla (f) The alternative method of cutting through the midportion of the frontal process of the maxilla is used only when the neoplasm is restricted to the inferior portion of the maxilla and therefore is rarely applicable

A sharp osteotome or a saw is used to cut through the zygomatic process of the maxilla (see Fig 4a) The periosteum is next elevated from the floor of the orbit in its anterior one half

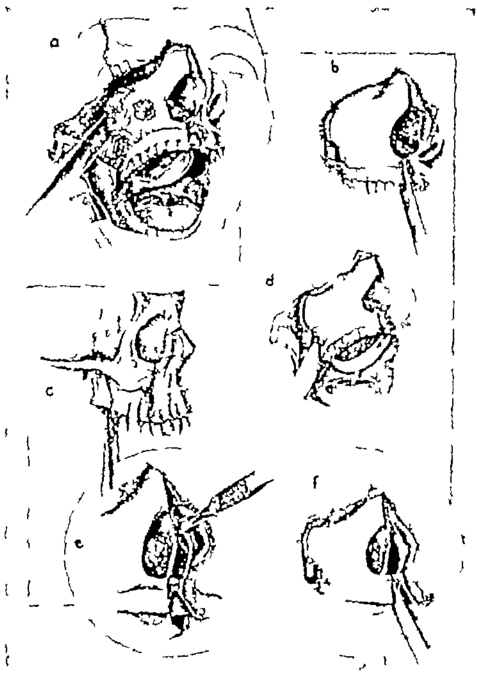

Fig 4 Cutting through the zygomatic process of the maxilla (b) Dividing the alveolus and palate near the midline (c) View of skull to show the relationship of the pterygoid processes to the posterior wall of the maxilla. (d) The osteome is placed behind the tuberosity of the maxilla and driven up into the pterygomaxillary fossa. (e) When the nasal septum and floor of the nose are invaded by neoplasm, the septum is divided anteriorly, thus exposing the opposite nasal meatus (f) The alveolus and palate are divided through the floor of the nose on the opposite side

The alveolus and bony palate are divided near the nasal crest with an osteome as shown in Fig. 4b.

The posterior surface of the resected block must be separated from the sphenoid bone. The palatine bone, which becomes part of the specimen, is separated from the pterygoid plates by placing an osteotome behind the maxillary tuberosity and driving it upward into the pterygomaxillary fossa (see Fig. 4, c and d).

Should the floor of the nose or the nasal septum be invaded by cancer the block resection should include those structures. The nasal septum is incised vertically anterior to the lesion so as to expose the opposite nasal meatus. The nasal bones and the alveolus and palate are divided in the manner described before, but on the opposite side of the midline (see Fig. 4, e and f).

The remaining areas of bony attachment usually break spontaneously. If they do not, this can be accomplished by gentle rocking of the maxilla. A fracture occurs through the orbital surface of the maxilla to the paper plate of the ethmoid. Sagging of the orbital contents will not occur because of the support of the zygomatic portion of the orbital floor and the intact orbital periosteum.

The palatine bone is wedged in between the maxilla and sphenoid bones and is firmly attached to the body of the sphenoid bone; therefore, the separation of the resected block of tissue from its posterior attachment usually occurs by fracture through the palatine bone. Thus the upper portion of the palatine bone remains attached to the anterior surface of the body of the sphenoid.

When the block has been loosened slightly, a narrow osteotome may be introduced through the cut in the nasal bone and carried posteriorly through the ethmoid cells, on the under surface of the orbital plate of the frontal bone. On reaching the anterior surface of the body of the sphenoid, the entire mass is displaced downward. When the nasal septum has been included in the resection, the perpendicular plate of the ethmoid is also divided along its superior attachment. A finger may be introduced into the nose to aid in delivering the resected block of tissue, as shown in Fig. 5a.

To control bleeding, hot towels are applied with pressure. The internal maxillary artery is grasped and tied. The smaller vessels may be coagulated. Remaining fragments of the ethmoidal labyrinth are curetted away. The sphenoidal sinus may be evacuated after removing its anterior wall. The final surgical defect is seen in Fig. 5b.

To aid in preventing the implantation of cancer cells, the area should be thoroughly irrigated with saline and the coagulating current used on all denuded areas.

Follow-up examinations for cancer of the maxilla and nose cannot be made satisfactory through the nostril or through a small oral opening. A large infralabial window must be constructed and kept open with a prosthetic appliance.

The window may be constructed in the following manner:

The labial mucosa is undermined and transfixed upward (see Fig 5, c and d). The anterior margin of the palatal flap is inspected for viability. The portion of this flap which was elevated from the alveolar process is thick and avascular and may have to be sacrificed. The anterior margin of the palate can *be folded upon itself and sutured down* (see *Fig.* 5, *d* and *e*). A palatal flap so constructed provides a pliable rim of soft tissue which allows easy introduction and removal of a prosthetic appliance without sacrificing its stability. Only the posterior end of the oral incision is sutured, so as to leave a window at least 4 cm. long. Gelfoam strips are laid on all raw surfaces. The cavity is packed with vaseline gauze strips in a manner that will allow easy removal from the oral window or nostrils. The subcutaneous layers of muscle and fascia are brought together with interrupted absorbable sutures. The skin is closed with fine silk sutures. The vermilion border of the lip should be accurately approximated (see Fig. 5f).

A head dressing is applied with moderate pressure over the operated area. Antibiotic and chemotherapeutic agents are used in doses sufficient to insure adequate blood levels for five to 10 days. The outside dressing is changed daily. The packing is left undisturbed for five days unless the patient develops an elevated temperature; in that case it must be removed earlier. The sutures may be removed on the fifth or sixth postoperative day. The cavity should be irrigated twice daily with warm, normal saline solution after the fifth day.

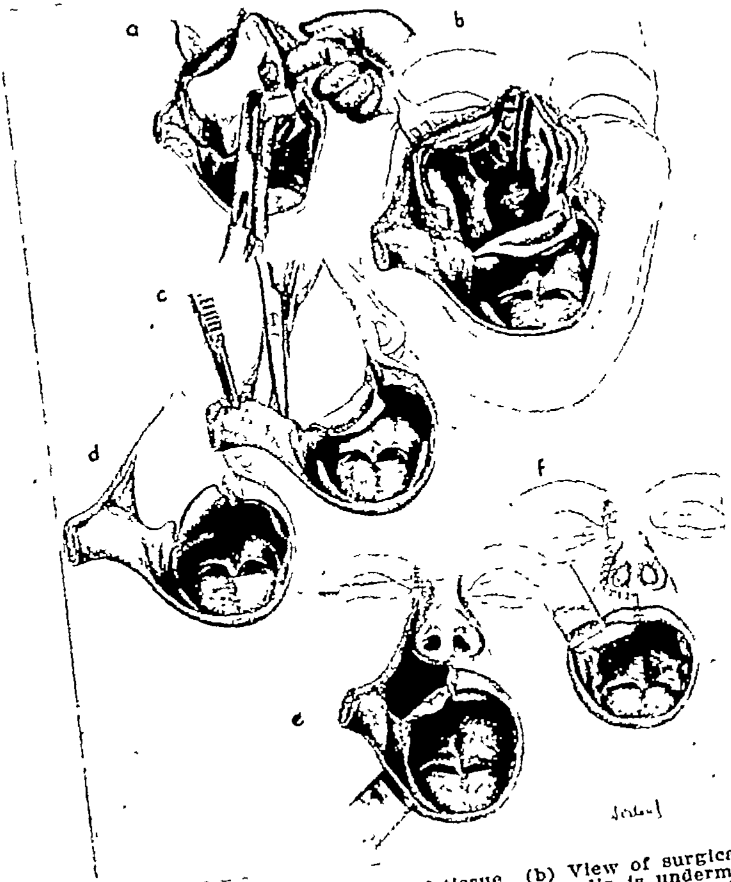

Fig. 5. (a) Delivering the block of tissue. (b) View of surgical d
(c and d) The mucosa on the inner surface of the lip is undermine
transfixed upward. (e) The anterior margin of the palate is folded o
itself and sutured down. (f) Closure completed, leaving a large infra
fenestra.

A prosthesis is constructed as soon as the gauze packing is removed. The appliance serves the following functions: *1.* It fills the infralabial opening and prevents contamination of the operative cavity with food and secretions. *2.* It prevents cicatricial stenoses of the window. *3.* It prevents deformity of the face by supporting the lip, nasolabial angle and cheek, and *4.* it provides a masticating surface (see Fig. 6).

When it has been possible to save a mucoperiosteal flap from the under surface of the hard palate, an elastic rim of tissue is provided along the medial and posterior margins of the window. Thus, the bulb of the prosthesis is constructed with a little overhang so that when it is introduced it will snap into place and thus be held firmly.[13]

The patient may leave the hospital between the seventh and tenth postoperative day. After two to three weeks all patients should receive intracavity radium or deep X-ray therapy. The patient continues to irrigate the cavity twice daily with normal saline solution until crusting and discharge have ceased, after which the irrigations are necessary once daily for an indefinite period. The patient should return for careful cleansing of the area at biweekly or weekly intervals over a period of two or three months until epithelialization of the cavity has occurred.

BLOCK RESECTION OF THE MAXILLA, ZYGOMA, AND CONTENTS OF ORBIT.

Proptosis almost always means that invasion of the orbit has occurred and, therefore, proptosis is an indication for evisceration of the orbit. The operation leaves a serious cosmetic defect. It should be recommended only after careful consideration of the patient's psychological attitude and after evaluation of the chances for a cure. Often the patient cannot carry on a gainful occupation after this operation. Most of these patients are elderly individuals, however, who are prepared to retire from extensive occupational and social contact with the public.

In this operation the face splitting incision is used, as already described, plus an extension carried laterally around the palpebral fissure about 5 mm. from the lid margins and for a distance over the zygoma, as shown in Fig. 7a. The

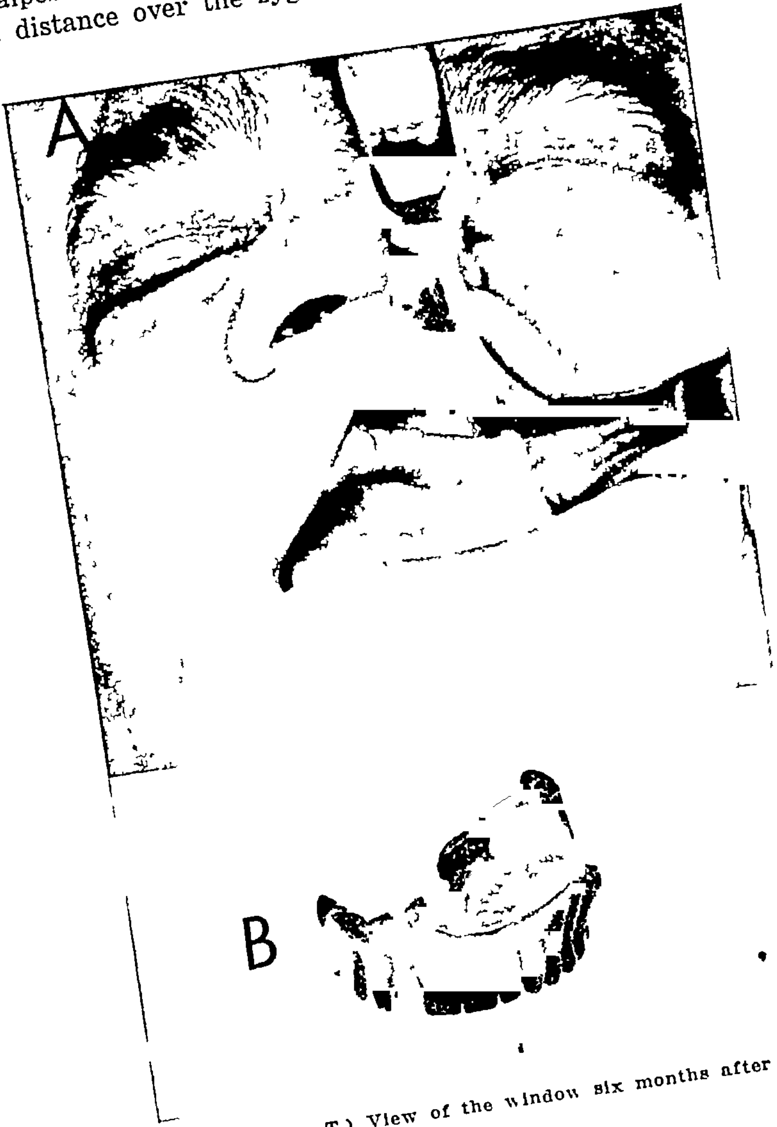

Fig 6 (a) (Mrs T) View of the window six months after op
(b) The prosthetic appliance

facial flap is reflected, as described previously. The skin of the upper lid is retracted to the supraorbital ridge. The zygomatic bone and the orbital contents become part of the resected block in this operation. The osteotome is used to break through the temporal and frontal processes of the zygoma. The maxilla, ethmoid, labyrinth, palate and nasal septum are handled in the manner previously described. The periosteum is reflected down from the roof of the orbit and the orbital contents are displaced downward until the pedicle of the eye is exposed. The pedicle is the last attachment to be cut. It is severed with a curved scissors, and the bleeding is controlled temporarily with hot packs and pressure until the ophthalmic artery can be grasped and tied (see Fig. 7b).

The anterior and inferior walls of the frontal sinus and the supraorbital ridge may be removed as shown in Fig. 7c. Any sharply protruding bone is cut away with a bone rongeur and the bone edges are rounded. The denuded surfaces are cauterized thoroughly. The upper lid is laid as a flap onto the roof of the orbit.

The mucosal border on the inner surface of the lip is sutured to the free edge of the palate so as to completely close the mouth from the operative cavity. The facial flap is laid onto the floor of the operative defect and its free margin sutured to the upper surface of the soft palate. The lip incision is closed to the lateral margin of the nostril. The part of the incision which extended over the zygoma is closed. The completed operation is seen in Fig. 7d. No attempt is made to close the surgical defect because *1.* primary closure usually breaks down and *2.* the presence of a large permanent opening facilitates both after-care and administration of radiation therapy. The packing, dressings and after-care is similar to that already described for resection of the maxilla.

Intracavitary radium therapy or deep X-ray therapy should be given after two to six weeks. Radium therapy can be given, incorporating radium needles into a wax impression of the operative defect.

After two to three months a prosthesis may be made t cover the surgical defect. The appliance should incorporate glass eye and restore the original appearance of the patient as far as possible. It can be made of latex rubber or some other lightweight material. Spectacles help to hold the prosthesis in place. The patient will not wear the appliance if it

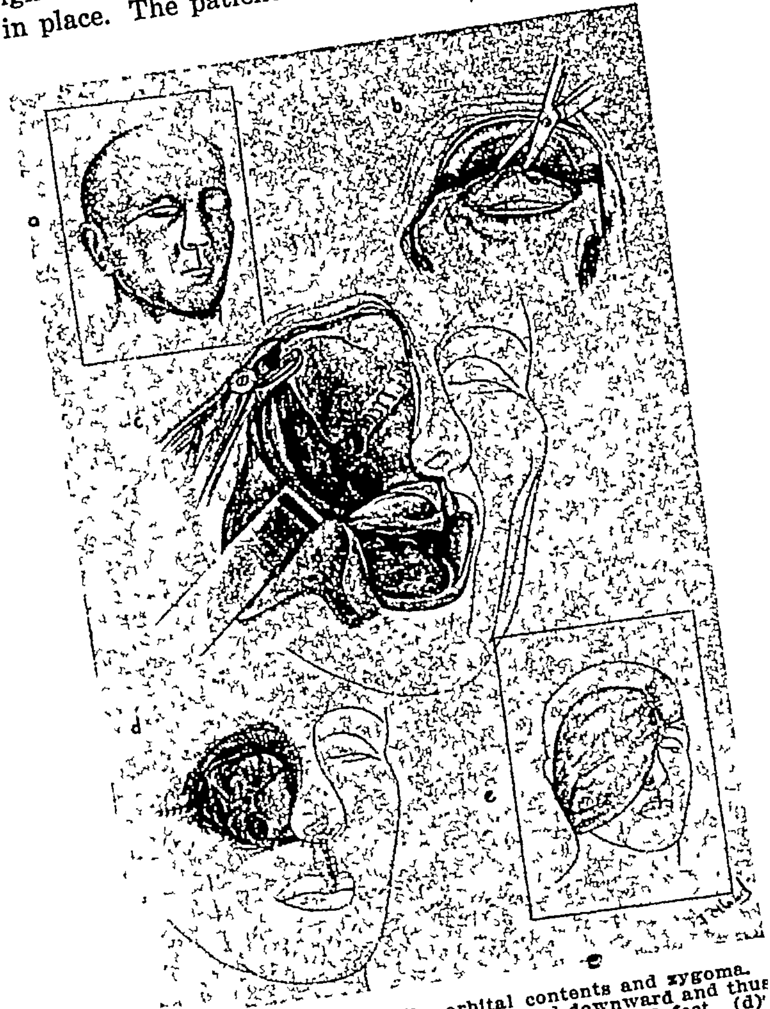

Fig 7 Resection of the maxilla, orbital contents and zygoma. incision. (b) The orbital contents are displaced downward and thus part of the resected block of tissue (c) The surgical defect. (d) completion of operation (e) Pressure dressing

is heavy, ill fitting or uncomfortable. Some patients prefer to wear a surgical dressing to cover the defect even though the prosthedontist has used the utmost care in constructing a good appliance.

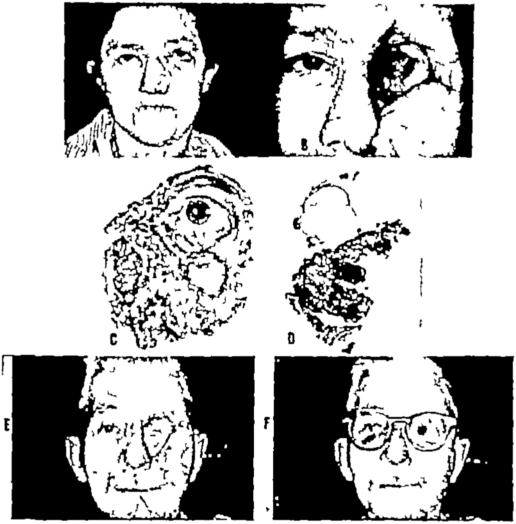

Fig 8 (Mrs. B) Left-sided proptosis indicates encroachment on orbital contents. (b) View of surgical defect on the fifth postoperative day (c) The resected block of tissue (d) Microscopic section through part of the specimen to show destruction of the orbital floor and displacement of the globe (e) (Mr R.) Surgical defect four months postoperatively (f) With facial prosthesis.

SUMMARY.

In general, the most effective treatment for carcinoma of the paranasal sinuses consists of surgical resection followed by radiation. Exceptions are those patients with radiosensitive tumors and those having malignancies beyond any reasonable chance for complete removal.

When block resection of the maxilla, ethmoid labyrinth and nasal septum is performed, a large permanent infralabial window should be constructed. A properly made prosthetic appliance will keep this window open and will prevent deformity by supporting the tissues of the face.

If proptosis has existed, the orbital contents must be included in the resection. This operation leaves an extensive facial defect. Many patients will accept the deformity when they understand that it is necessary for a cure.

Radiation therapy should routinely follow all surgical resections for carcinoma of the paranasal sinuses.

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THE ARTICULATION FUNCTION
OF
PATIENTS WITH CONDUCTIVE DEAFNESS.*

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INTRODUCTION.

Otologists have long recognized that certain hard of hearing patients understand speech with relative ease when it is made loud enough to override their hearing losses. Other patients complain that they "hear" speech but do not understand it, and are helped more by slow delivery and careful enunciation than by mere increase in intensity. From these observations, the concept of two related yet distinctly different dimensions of hearing for speech has emerged. The first dimension, sensitivity, is the ability to detect soft speech. The second, discrimination, is the ability to understand audible speech by differentiating between phonetic elements. A test of sensitivity, therefore, tells us how intense speech must be made *before the patient responds to it, while a test of discrimination* tells us how well he recognizes words in speech which is already loud enough. In general, a simple reduction of sensitivity, a so-called "hearing-loss", has been associated with conductive deafness, while difficulty in discrimination has been related to nerve deafness.

Word lists for testing hearing for speech were compiled during the war at the Psycho-Acoustic Laboratory of Harvard

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*From the Hofheimer Audiology Laboratory, Department of Otolaryngology, Washington University, and Central Institute for the Deaf. This work was begun under contract Noonr-272 between the Office of Naval Research and Central Institute for the Deaf. It was continued under contract V1001M-577 between Veterans Administration and Central Institute for the Deaf, later transferred to the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University). The work there is supported by a grant from the Hofheimer Foundation.

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University.[1] Spondaic (disyllabic) words such as "railroad" and "stairway" were chosen, since they were found to be of high and approximately equal intelligibility. A test of discrimination, on the other hand, should present the difficulties encountered in understanding everyday English. The speech which we hear varies in intelligibility partly because some phonetic elements are harder to identify than others. The phonetic composition of English was therefore taken as the basis for construction of this test material. Twenty "phonetically balanced" lists of 50 monosyllables each were compiled at the Psycho-Acoustic Laboratory,[1] each list containing the phonetic elements in approximately the proportion in which they occur in English.

The general procedure in testing sensitivity with spondaic words is to attenuate the stimulus until a level is determined at which the patient is able to repeat 50 per cent of the words correctly. This level is called his "threshold for speech" and when compared with a threshold for normals obtained under the same conditions, provides a measure of loss in sensitivity in decibels. In testing discrimination, a list of monsyllables is presented at a constant intensity, and the patient's "score" is the percentage of words repeated correctly at that level.

If a series of phonetically balanced ("PB") word lists is given to a patient at intensity levels successively farther above his threshold for speech, his scores may be plotted against physical intensity (in decibels). As the intensity is increased he understands more and more of the words, until, with sufficient intensity, the test material becomes maximally intelligible for him. Such a curve is called an "articulation function".[1] In 1946, Walsh and Silverman[2] advanced the theory that the shape of the articulation function for the hard of hearing person depends upon the type of his hearing loss. They published sample articulation functions obtained on three patients whose hearing losses were, respectively, of the conductive, mixed and nerve types. At intensity levels well above threshold, the patient with conductive deafness could repeat nearly all the PB words correctly, but the patients with nerve degeneration could not. Walsh and Silverman suggested that such articulation tests may, therefore, be useful in diagnosis and prognosis for the fenestration operation.[cf. also 8]

The intelligibility of speech depends not only on its loudness and phonetic content, but also on the voice and enunciation of the speaker. No two speakers say a word in exactly the same way, and one speaker does not produce identical acoustic patterns as he repeats the same word. In many hearing clinics hearing for speech is tested by live voice. At Central Institute for the Deaf, however, in order to obtain standardized results, recordings of eight of the phonetically balanced word lists were made by Harrison in the Technisonic Laboratory at Central Institute for the Deaf, in 1945.[4] The talker was Rush Hughes, a professional radio announcer. These recordings, although made originally as a temporary expedient to test the advantages of recorded lists, have been used at Central Institute for the Deaf since October, 1945 and subsequently in the Hofheimer Audiology Laboratory at McMillan Hospital (Washington University) to test the auditory discrimination of all patients of Dr. T. E. Walsh who were candidates for the fenestration operation.

A "normal" articulation function for the PB lists is given by Walsh and Silverman (Figs. 5 and 7). These curves were only tentative, and the strong concavity upward in the lower part of the curve was not found in a more extensive study two years later (Davis, "Articulation Area", Fig. 8). Even this function rests on a rather small foundation, namely a group of ten normal subjects. The experiments were not systematically counterbalanced to eliminate learning factors and it was assumed (a) that all eight recorded lists were equally difficult and (b) that the normal function would reach 100 per cent correct at the level of average speech. In drawing the "normal" curve in question[2] we took into account the extensive data obtained at the Psycho-Acoustic Laboratory with the same lists of words.[1] The work at Psycho-Acoustic Laboratory, however, was done with live voice, not with our recorded tests. We have not studied the normal articulation curve for the Rush Hughes recording in further detail because we have always regarded these particular recordings as temporary stop-gap items, but only recently have we started serious work on a new set of recordings to replace them. In the meantime other laboratories have obtained and employed copies of the Rush Hughes recordings. For them the present data will have a practical as well as a theoretical interest.

A somewhat similar study has recently been published by Beasley and Rosenwasser.[5] They determined separately the articulation functions for two sets of (recorded) monosyllables. One set is composed of "high-frequency words", the other of "low-frequency words". The two articulation functions were determined for 50 normal ears. The two functions were almost identical in shape (as well as in threshold value), and the authors state that "a composite average between the two curves does not differ significantly from the normal articulation curves for PB word lists as reported by Davis[6] and Thurlow.[7] The coincidence is gratifying, even though the word lists were not identical with our own. Incidentally, the lower levels of the plateaus (or maxima) of the articulation functions found by Beasley and Rosenwasser for patients with hearing losses (particularly high tone hearing losses) agree very closely with our own experience.

METHOD.

The group of patients and the routine of testing have been described in full elsewhere.[7,8,9] Articulation tests were conducted systematically both before and after the fenestration operation and on the unoperated as well as the operated ears. Nearly all of our articulation scores for the Rush Hughes recordings obtained on patients fenestrated without stopple enter into the present study. The series of operations extends from January, 1945 to July, 1949. They are consecutive cases except for "revisions" and also excepting four cases for whom Spanish was the mother tongue and two who failed to cooperate because of psychological difficulties.

The clinical diagnosis of nearly all of these patients was "otosclerosis". The degree of nerve involvement varied from none (pure conductive hearing loss) to moderate (mixed deafness, predominantly conductive or "conductive-mixed") to considerable (mixed deafness, predominantly perceptive or "mixed-nerve"). No cases of pure nerve deafness have been fenestrated, and this important group is, therefore, excluded from the present study. The classification was made before the operation by Walsh on the basis of all available evidence, including history, tuning fork tests, pure tone audiometry and (in the last three years) tests of discrimination for speech. In only a very few cases was it necessary to revise the classification on the basis of additional information obtained at the operating table.

In each testing session the threshold for speech was measured by Auditory Test No. 9,[10,7] a recording of spondaic words originally prepared at the Psycho-Acoustic Laboratory of Harvard University and transcribed in the Technisonic Recording Laboratory.[4] The threshold for speech determined by Test No. 9 is the common reference point for all cases. The intensities at which the Rush Hughes PB lists were given are all reckoned as decibels above the threshold (for that ear) for Test No. 9. The hearing losses for speech range from about 20 to about 80 db.

Our routine for giving the PB tests has been changed several times. Early in the program, PB lists were given at intensity levels 10 db apart, from a level near the patient's threshold (for Auditory Test No. 9) to a high level such as 110 or 120 db re .0002 dyne/cm^2. In this way, a complete articulation function was obtained for each ear tested. Subsequently the routine was changed to include only three lists, two given at least 35 db above the threshold for Auditory Test No. 9 and one given at the patient's threshold of intelligibility for connected discourse.[11] For the latter, we later substituted a test at 85 db re .0002 dyne/cm^2 (the level of loud speech). The relationship between this intensity and the patients' thresholds for Auditory Test No. 9 varied, of course, with the hearing loss of individual patients. This procedure was followed for about one year. During the past two years, the PB test has been given only at levels at least 35 db above the threshold for Test No. 9. By changing our routine as described above, we have given PB tests at every intensity level relative to the threshold for Auditory Test No. 9, from a few decibels below to about 60 decibels above.

Now, after five years of practical experience with these tests, we have examined the scores systematically in order to answer the following questions:

1. Are the articulation scores of the hard of hearing (using the Rush Hughes recording) correlated with clinical diagnosis, as suggested by Walsh and Silverman?
2. Are the eight Rush Hughes lists of equal difficulty?
3. What is the distribution of difficulty among individual test items in the Rush Hughes recording?
4. How reliable is the maximum articulation score ("PB-Max")?

The study indicates certain specific shortcomings of the Rush Hughes recording which should be avoided in any recordings of PB lists to be prepared in the future.

RESULTS.

The composite articulation functions (see Figure 1) show the mean PB scores, averaged at intensity levels 5 db apart, from the threshold for Auditory Test No. 9 to 55 db above it. The number of scores averaged to obtain each point on the curves was considerable (see Table 1). Actually, each point represents a range in input level of 2.5 db above and below each indicated level; that is, scores on tests given from 2.5 db below to 2.5 db above the threshold for Test No. 9 were averaged to obtain the mean score at the threshold for Test No. 9; tests given from 2.5 to 7.5 db above threshold were used for the point 5 db above the threshold for Test No. 9, and so on.

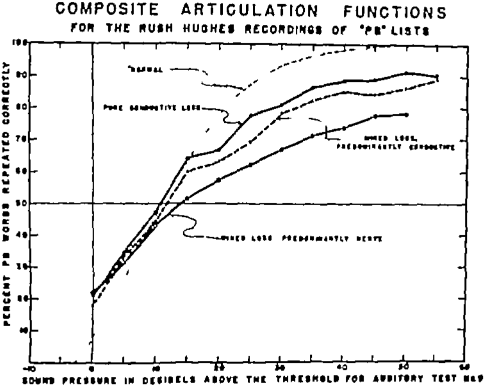

Fig 1

TABLE I

ARTICULATION SCORES OF PATIENTS WITH PURELY CONDUCTIVE HEARING LOSS

Input Level Db Above No 9 Threshold	No of Tests	No of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score ±σ	Range of Scores (Per Cent Correct)
0	97	48	21.3	12 8	1.26	8 9-33 6	0- 48
5	74	46	34.5	13 0	1 51	21.5-47.5	4- 72
10	93	41	47.1	14.1	1 47	33 0-61.2	4 84
15	81	47	64 0	14 0	1.56	50 0-78 0	30- 90
20	84	38	66 8	16 8	1.83	50 0-83 6	42- 92
25	78	45	77 6	9.9	1 12	67 7-87.5	38 96
30	118	54	81.0	8 4	.77	72 6-89 4	56- 98
35	229	93	86 6	7 1	47	79 5-93 7	64 100
40	364	115	88.7	6.3	.33	82.4 95 0	64 100
45	254	87	89.1	6.2	39	82.9-95.3	70-100
50	263	79	91.3	5 8	.36	85.5 97.1	70-100
55	155	57	90 6	6 4	.52	84.2 97 0	72-100

*σ = standard deviation of the mean.
σm = standard error of the mean.

Correlation with Clinical Diagnosis

Near the threshold for Auditory Test No. 9, the performance of the groups with and without some nerve deafness is very similar. The mean scores are *not* significantly different (by the t-test)* at the threshold for Test No. 9 or at 5 or at 10 db above it. At the threshold for Test No. 9, where all groups make about 20 per cent, the mean for the mixed nerve cases is actually highest, and the point at which 50 per cent of the PB words are heard correctly (the "PB threshold") is passed by all three groups between 10 and 15 db above the threshold for Test No. 9. It seems, therefore, that PB tests given at levels near the threshold for spondaic words do not

TABLE II.

ARTICULATION SCORES OF PATIENTS WITH MIXED HEARING LOSS, PREDOMINANTLY CONDUCTIVE.

Input Level Db Above No. 9 Threshold	No. of Tests	No. of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score ±σ	Range of Scores (Per Cent Correct)
0	26	16	18.0	13.7	2.74	4.3-31.7	0- 50
5	45	22	33.5	14.3	2.13	19.2-47.8	4- 70
10	39	22	44.3	16.3	2.63	28.0-60.6	16- 76
15	32	20	60.0	10.5	1.84	49.5-70.5	32- 80
20	38	19	63.1	12.3	1.98	50.8-75.4	32- 88
25	35	17	69.4	11.0	1.86	58.4-80.4	42- 86
30	65	30	78.3	7.7	.95	70.6-86.0	58- 94
35	111	37	82.6	8.5	.81	74.1-91.1	60-100
40	149	45	85.3	6.8	.56	78.5-92.1	64- 98
45	101	38	84.5	6.9	.69	77.6-91.4	62- 98
50	124	36	86.8	6.8	.61	80.0-93.6	68- 98
55	47	21	89.2	5.9	.86	83.3-95.1	76-100

*σ = standard deviation of the mean.
σm = standard error of the mean.

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*The t-test measures the probability that the difference between two means is the result of random factors in sampling.

indicate the presence or absence of nerve involvement. Evidently the easiest PB items are just as intelligible as the spondaic words of Test No. 9, whether or not the listener has some nerve deafness. The critical items for discrimination are the 25 hardest words, and the test is most useful for this purpose when given at levels well above the threshold.

TABLE III.

ARTICULATION SCORES OF PATIENTS WITH MIXED HEARING LOSS, PREDOMINANTLY NERVE.

Input Level Db Above No 9 Threshold	No of Tests	No. of Patients	Mean Score (Per Cent Correct)	σ*	σm*	Mean Score ±σ	Range of Scores (Per Cent Correct)
0	43	24	21 8	11 4	1 73	10 4-33.2	0- 46
5	53	27	31 5	11 6	1 59	19 9-43 1	8 60
10	64	27	43.1	12 7	1 59	30 4-55 8	12- 74
15	60	27	51 7	10 8	1 40	40.9-62.5	22- 76
20	78	28	57 2	12 1	1.38	45.1-69.3	22 76
25	65	26	62.1	10 2	1 26	51.9-72.3	40- 82
30	79	27	67 1	10 1	1 13	57 0-77 2	38- 90
35	78	23	71 4	9 4	1 07	62.0-80 8	40- 90
40	75	22	74 0	9 9	1.14	64 1-83.9	64 88
45	39	15	77 7	13 8	2.23	63 9 91 5	60- 96
50	26	11	78.2	7 2	1 44	71 0-85 4	64- 90

*σ = standard deviation of the mean
σm = standard error of the mean

As the intensity is raised to 15 db or more above the threshold for Test No. 9, the curves diverge significantly. The means for patients with mixed-nerve loss are *significantly* lower than for those with conductive-mixed loss at all intensities 15 db or more above the threshold for Test No. 9. The level of confidence that this is a real difference is between .01 and .001, or better, except at 20 db above the threshold for Test No. 9, where it is .02. (The fraction .01 indicates that the probability is not more than one in a hundred that the result found could have occurred simply because of random differences. The fraction .001 indicates that the probability is only one in a thousand, and so on). The conductive-mixed curve diverges *significantly* from the pure-conductive curve at 25 db above the threshold for Test No. 9. From 25 to 50 db above the threshold for Test No. 9, the means for conductive-mixed loss are *significantly* lower than those for pure-conductive loss. (The level of confidence is better than .001, with the exceptions that at 30 db above the threshold for Test No. 9 the level of confidence lies between .05 and .02 and no significant difference was found at 55 db above the threshold for Test No. 9.) At levels well above the threshold for Test No. 9, therefore, the group of patients with nerve involvement gives a significantly inferior performance on the Rush Hughes recording, and the greater the nerve involvement, the nearer threshold the inferior performance shows up. The data thus confirm the theory of Walsh and Silverman that performance on the PB test is correlated with clinical diagnosis.

Maximum Articulation Scores

In clinical practice, we have considered it most useful to give the PB test at an intensity level where the patient will be able to give his maximum performance, in other words to attempt to measure the highest point on his articulation function. The question arises of how far above the threshold for Test No. 9 the intensity should be set in order to measure the maximum PB articulation score, or "PB-Max". The articulation functions never reach 100 per cent, and their upper portions show a very gradual but significant improvement in score with increasing intensity, rather than a well-defined plateau. The means (for patients with pure-conductive loss) at 50 and 55 db above the threshold for Test No. 9 are *significantly* higher than the mean at 40 db above. (The level of confidence for this difference is better than .001). It is clear that to measure the maximum articulation score, the test should be given at least 40 db above the threshold for Test No. 9. It is not always practical, however, to give the test at such a high level, particularly to patients with considerable hearing loss, as the required level may be above the threshold of discomfort; but if the test is given at lower levels, a correction can be applied to the measured score in order to obtain a better estimate of the true maximum value. Table IV lists the number of percentage points that should be added to the observed score according to the level at which the test is actually administered. These corrections are based on the shape of the average articulation function for all of the patients, including those with some nerve deafness as well as those with purely conductive deafness.

TABLE IV.

CORRECTION FOR RUSH HUGHES "PB-MAX" SCORES OBTAINED BELOW THE TRUE MAXIMUM

Level Decibels Above Threshold for Test No 9	Correction Percentage Points; Add to Score
25	14
30	8
35	4
40	1
45	0

The differences between the mean "PB-Max" scores for the three diagnostic groups were determined by averaging for each group separately all of the tests given from 40 to 55 db above the threshold for Test No. 9. The results are shown in Table V. The differences between the mean "PB-Max" scores are *significant* at a level of confidence better than .001.

TABLE V.

MAXIMUM PB ARTICULATION SCORES.

Diagnosis	No of Tests	Mean Score (Per Cent Correct)	σ	Mean Score $\pm\sigma$
Pure Conductive	1036	89.7	6.2	83.5 to 95.9
Conductive Mixed	421	86.0	6.8	79.2 to 92.8
Mixed Nerve	140	75.8	10.8	65.0 to 86.6

Our test differentiates most clearly between pure-conductive and mixed-nerve loss. There is very little overlap between the ranges of plus and minus one standard deviation around the means for these two groups. The bottom of this range for pure conductive loss is 83.5 per cent, and the top for mixed nerve is 86.6 per cent. Eighty-five per cent, the center of this small area of overlap, may be taken as a practical dividing line between scores showing good discrimination and those showing impaired discrimination.

The percentages of the total number of "PB-Max" tests which fall into various score ranges is useful in the interpretation of individual tests. This information is given in Table VI.

TABLE VI.

DISTRIBUTION OF MAXIMUM ARTICULATION SCORES.

"PB-Max" Score (Per Cent Correct)	PURE CONDUCTIVE		CONDUCTIVE-MIXED		MIXED-NERVE	
	No. of Tests	Per Cent of Tests	No. of Tests	Per Cent of Tests	No. of Tests	Per Cent of Tests
90 to 100	616	59.5	135	32.1	5	3.6
80 to 88	359	34.6	221	52.5	53	37.8
70 to 78	59	5.7	60	14.2	56	40.0
68 and below	2	0.2	5	1.2	26	18.6
TOTAL	1036	100.0	421	100.0	140	100.0

Since the mean score for pure-conductive loss is just under 90 per cent, it appears that a score of 90 per cent or higher on the Rush Hughes test is excellent. Seventy-eight per cent, on the other hand, is a poor score, strongly indicating nerve involvement. Scores from 80 per cent to 88 per cent are inconclusive, since patients in all three groups score in this range.

Reliability

There is considerable dispersion of scores around the means in all parts of the articulation function. The standard devia-

Relative Difficulty of Rush Hughes PB Lists

Three studies have been carried out to measure differences in difficulty between the eight Rush Hughes PB lists. The first consisted of grouping the "PB-Max" scores of our pure conductive cases according to list and comparing the mean score for every list with that of every other list by means of the t-test. In the second study, the same procedure was followed using the "PB-Max" scores of the conductive-mixed group. The third study was a planned comparison of lists carried out on consecutive patients as they came to McMillan Hospital for testing, without selecting the subjects on the basis of diagnosis. In this study the lists were paired systematically and the maximum articulation score was determined with each pair for each of twenty hard-of-hearing ears. The intensity of the two experimental lists was the same for each ear tested, and the sequence of the two lists was varied from patient to patient to counterbalance fatigue and learning effects. The method of pairing was as follows: Lists 6 and 12 were used as reference lists; List 6 was first paired with Lists 5, 7 and 8; then List 12 was paired with Lists 9, 10 and 11; and finally Lists 6 and 12 were compared. In this way, the eight lists could be arranged in order of difficulty on the basis of average differences in score.

The order of difficulty of lists, from easiest to hardest, as indicated by the three studies, is as follows:

All Conductive cases	11, 12, 10, 7, 5, 9, 6, 8
All Conductive-Mixed cases	12, 11, 10, 7, 9, 6, 5, 8
Planned Comparison series	12, 11, 9, 7, 5, 10, 6, 8

In all three studies, Lists 11 and 12 were found to be easiest, and List 8 hardest. The statistical evidence of this was more striking in the conductive than in the conductive-mixed study. For patients with pure conductive loss, List 8 was significantly harder (by the t-test, level of confidence .05 or better) than every other list, while for the conductive-mixed group it was significantly harder than four other lists. List 11 was significantly easier than six other lists in the conductive study, but it was significantly easier than only three lists in the conductive-mixed study. Perhaps the reason for the more uniform difficulty of the lists for the conductive-mixed group is that patients with some nerve involvement make lower scores. They, therefore, make mistakes not only on the five or six most difficult items, but also on the ten or twelve most difficult. As more items become critical, the lists apparently become more nearly equivalent.

List 11 is easier than List 12 (for the conductive group) probably because it contains no extremely difficult items, while List 12 includes the words "frill" and "greet" which are very frequently missed. More patients are, therefore, able to make 100 per cent on List 11. Of our group of 1036 "PB-Max" scores on pure conductive cases, there were 27 perfect scores, 14 of which were made on List 11.

A detailed study of the significance of the differences in difficulty among the five other lists in the three studies has led us to the general conclusion that Lists 5, 6, 7, 9 and 10 are of practically equal difficulty. Such significant differences as appeared in one study were not confirmed in the other two. List 8 is, however, *significantly* harder and Lists 11 and 12 are *significantly* easier than the other five. The differences in average "PB-Max" scores for the different lists are of the order of only 2 per cent.

If the Rush Hughes recordings of Lists 8, 11 and 12 are to be employed for determining the maximum articulation scores of patients with conductive or mixed hearing losses, the following corrections should be applied:

List 8:	add 2 percentage points to the apparent "PB-Max" score
Lists 11 and 12:	subtract 2 points from the apparent "PB-Max" score.

These corrections are to be applied in addition to the correction for intensity given in Table IV. The corrections are small, but they are important because the differences between the mean "PB-Max" scores of the diagnostic groups are not large.

DISCUSSION.

We formerly believed[2,3] that the articulation function for patients with pure conductive hearing loss was a simple displacement on the intensity scale of the normal articulation function; however, the curve which we have now constructed for the conductively deaf differs from the old "normal" curve in three respects. The score at the level of the threshold for Auditory Test No. 9 is about 20 per cent, not 8 per cent; the curve is less steep; and it never reaches 100 per cent. It continues to rise gradually and significantly at levels as much as 50 db above the threshold for Test No. 9, but it never gets much higher than 90 per cent. Just how significant these differences from normal are we cannot tell until data are available on a large group of normal but untrained listeners.

The data show several characteristics of the Rush Hughes recording which might be improved upon in any new recordings to be made in the future. The wide range of difficulty among individual test items is probably not ideal. It is true that, in a clinical test, the easiest items should be easy enough to make the test practical for testing patients with very poor discrimination, and the most difficult items should be hard enough to make the test sensitive to small differences in discriminatory ability found among normals and among patients with pure conductive hearing loss; but the most difficult items must not be so hard that the test becomes unrealistic. The range of difficulty in the Rush Hughes recording may be too wide; also, it would be a practical advantage in a clinical test if a definite plateau were reached at 35 db above the threshold for spondaic words. It would then more often be possible to measure the maximum articulation score directly without using a correction.

There is evidence that the distribution of difficulty among items in the Rush Hughes recordings is uneven. We have performed an item analysis of the errors made by conductive-deaf patients on three of the eight individual lists. The analysis was confined to the tests given 40 to 55 db above the threshold for Test No. 9. It was obvious that certain words such as "flip", "spice" and "deuce" in List No. 8 were particularly difficult and that the other lists contained fewer or no items of comparable difficulty.

There is an irregularity at the 20 db level in the shape of the average articulation functions for all lists (see Fig. 1). This depression is significant for the pure-conductive and for the conductive-mixed groups. We believe that this irregularity is a spurious characteristic of the Rush Hughes recordings.

We hope that our experience with the Rush Hughes recordings will prove valuable to those who attempt to produce better recorded versions of the PB lists in the future.

SUMMARY.

1. A set of recordings by Rush Hughes of eight "phonetically balanced" word lists has been used for five years at Central Institute for the Deaf and in the Hofheimer Audiology Laboratory (McMillan Hospital) to test the auditory discrimination of candidates for the fenestration operation. From the data which have been collected, composite articulation functions for the Rush Hughes recordings, based on the threshold for Auditory Test No. 9, have been constructed for three groups of patients, diagnosed respectively as follows: (a) pure conductive loss; (b) mixed loss, predominantly conductive; and (c) mixed loss, predominantly nerve. In averaging the articulation scores of different patients, each patient's threshold for Auditory Test No. 9 was taken as the reference point.

2. Comparisons by the t-test of the mean scores of the three clinical groups at levels from the threshold for Auditory Test No. 9 to 55 db above it show that, at levels 25 db and higher above the threshold for Auditory Test No. 9, performance on the Rush Hughes recordings of PB lists is correlated with clinical diagnosis, as suggested by Walsh and Silverman in 1946.

3. Since the articulation functions are still rising gradually but significantly 40 and 45 db above the threshold for Test No. 9, the intensity chosen for measurement of a patient's maximum PB articulation score should be as far above his threshold for Auditory Test No. 9 as is practical, if his hearing loss is wholly or partially conductive.

4. The articulation functions indicate that the range of difficulty among individual test items in the Rush Hughes recordings is very wide and that the distribution of difficulty is uneven.

5. The reliability of the Rush Hughes test is not very high. At levels 40 to 55 db above the threshold for Auditory Test No. 9, the standard deviations around the mean scores never become much lower than 6 per cent.

6. Comparisons of the eight Rush Hughes PB lists show that List 8 is significantly hard, Lists 11 and 12 are significantly easy, and Lists 5, 6, 7, 9 and 10 fairly well equated in difficulty. Correction factors to be used with Lists 8, 11 and 12 are given.

7. Our experience with the Rush Hughes recording points out certain problems which must be considered when new recordings of PB lists are made.

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THE TONGUE AS A FOCAL INFECTION.

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In this age of exact scientific method, bacteriologists, pathologists and internists are extremely skeptical of proof obtained by clinical evidence alone. The doctrine of focal infection has not been completely proved by scientific methods and, therefore, has become quite controversial. As a matter of fact, judging by recent publications, its pendulum has swung pretty low, nor is this paper to hold up the tide against it. Verhoef[1] states, "Belief in focal infection must be taken like religion — on faith." Slocum *et al.*,[2] of the Mayo Clinic, evaluate the concept of focal infection on clinical observation alone. In much the same way, I shall cite a few cases where the therapeutic response to the eradication of the focus of infection seems to justify this doctrine as a working hypothesis, if nothing more. Woods[3] states: "It is an established procedure with the ophthalmologist to search for foci of infection in the study of uveal tract diseases." Many other diseases of the eye and its appendages may also be cured by eliminating foci of infection. Thus in another article[4] written in 1932, I illustrated that dacryocystitis may be relieved by eliminating foci of infection. A very convincing clinical proof of focal infection is the following case:

Case 1: Female, aged 30, had a peritonsillar abscess in 1919 which was opened and drained. She was then advised to have her tonsils extirpated, but she refused. Four years later she had a fulminating optic neuritis in one eye with hemorrhages and exudates all around the papilla, which was not elevated. Upon examination of the upper respiratory tract, it was found that the tonsils were infected and two gold-capped teeth were present. X-ray of the teeth revealed no infection. The tonsils were removed and 48 hours later the eye cleared, and the vision returned to normal.

Four years later she had a similar attack in the same eye. She was examined thoroughly by an internist, had X-rays taken of all parts of the

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body, and was subjected to all other possible means of investigation. All tests were found to be *absolutely negative*. Three weeks were consumed in the examinations, and the patient did not get any better. It was only after an X-ray of the teeth that an abscess was found, which was eliminated by extraction. The eye cleared of the acute infection within 48 hours. It did not return to normal, however, being left with a partial secondary atrophy of the optic nerve.

Two years later she had another attack in the same eye. She was then edentulous; tonsils were extirpated with no residual tissues visible, and sinuses were clear on transillumination and X-ray. She volunteered the information that she had some gynecological disorder. She was sent to a gynecologist, who reported that she had a definite chronic endocervicitis and recommended cauterization. This having been done, in 48 hours the eye again cleared to the status preceding the last episode.

Against this clinical evidence grave objections may be raised, chiefly that patients do get well spontaneously and that some patients who had their tonsils removed, teeth extracted and perhaps sinuses punctured obtain no results, or are possibly made worse. With regard to the former point, Woods[7] claims that the clinical evidence for recovery on removal of foci of infection is so formidable that it is difficult to dismiss it lightly on the ground that it is only suggestive. With regard to the latter point, Slocum *et al.*[8] demonstrated that where the malady is irreversible or irreparable, removal of foci will be of no therapeutic benefit. On the other hand, can we honestly say that we have eradicated all foci of infection when we have eliminated a few obvious ones? Lederer[9] claims that the phrase "ear, nose and throat essentially negative" does not mean anything unless we have used all means at our disposal for examination, such as the laryngeal mirror to investigate the lingual tonsils, the nasopharyngoscope or a postnasal mirror for nasopharynx, transillumination and X-ray of the sinuses. Otherwise, we leave out many possible sources of infection.

Davis[4] reported cases which he had miraculously relieved of pain and remote disease by cauterizing some infected follicles of the lingual tonsils or lateral bands of the pharynx or follicles anywhere in the nasopharynx. He concludes by saying: "*1.* Tonsils do not constitute the only foci of infection in the pharunx. *2.* Other foci are lingual tonsils, adenoids and lymphoid tissue on the posterior pharyngeal wall."

There still remains another possible focus of infection in the oral cavity to which very little attention has been paid, and that is the tongue—particularly the coated tongue. Gray[7] states that the tongue presents a depression, the foramen cecum from which a shallow groove, the sulcus terminalis, runs lateralward and forward on either side to the margin of the tongue. The part of the dorsum of the tongue in front of this groove, forming about two-thirds of its surface, looks upward, and is rough and covered with papillae; the posterior third looks backward and is smoother, and contains numerous muciparous glands and a large number of lymph follicles, which together constitute what is sometimes termed the lingual tonsil. Elfman[8] claims that the lingual tonsils are compensatory to faucial tonsils and are similar in structure to them. They are subject to the same affections and may act as focal infection in the same manner as the faucial tonsils do. A striking example is the following case:

Case 2: C. E., female, aged 23 years, had a history of general glandular enlargement, duration of one year, accompanied by low-grade fever and malaise. All tests were made and proved negative, including complete hematological and serological tests and biopsy of one gland. Examination of the upper respiratory tract revealed enlargement of the lingual tonsils with follicles all over the posterior part of the tongue. These follicles from time to time became acutely inflamed. A higher elevation of temperature occurred with the acute inflammation. She was anesthetized, and the lingual tonsils were removed along with the surrounding lymphoid follicles. She made an uneventful recovery, swelling of all glands subsided, temperature became normal, and she has remained well ever since, a period of 12 years.

The papillae covering the anterior two-thirds of the tongue are minute surface eminences. They consist of a layer of connective tissue covered with epithelium. The filiform papillae are the most numerous, occupying the general area anterior to the large circumvallate papillae, which form the inverted V-shaped terminal sulcus at the base of the tongue.

Crohn and Drosd[9] state that each filiform papilla is surrounded by projections, the "secondary" papillae which protrude like flexible spines or spikes, covered by stratified squamous epithelium.

Henning[10] found the filiform papillae to be plump, round hillocks with transparent epithelium. Most characteristic were the secondary papillae which jutted out from the filiform papillae like blades of grass with serrations. When the tongue was coated the papillae were long, thick, firm and tense, and were surrounded by leucocytes, bacteria and particles of debris.

Bloomfield[11] demonstrated that bacteria grow normally not only on mucous membrane but also in their superficial layers, in crevices of the superficial layers and between them, and in orifices of small glands.

The coating of the tongue according to Boas[12] is ideal for the development of an offensive odor of putrefaction and calls it "fetor ex lingua." While fetor ex ore can be produced by many disorders, such as gastrointestinal diseases, lung abscesses, bad teeth and their surrounding tissues, infected sinuses, malignancies in the throat and mouth, there is sometimes an unique odor of putrefaction without any demonstrable malady; however, the tongue is usually coated in these cases and washings from it emanate the same characteristic odor. That this odor can be produced by the tongue alone, I have proved in an earlier paper[13] written in 1932, which was demonstrated with the following case:

Case 3: B. L., child, female, 12 years old, had been suffering from bad breath *for several years. Tonsils and adenoids were removed with no* beneficial effect. She was surveyed on two different occasions in two leading hospitals and nothing abnormal could be found, except a slight postnasal discharge, for which she was treated. The discharge when removed from the nasopharynx proved to be odorless. There was no bad taste complaint, the appetite was good, and the weight was normal. In spite of the fact that the gastrointestinal tract was found to function properly, her tongue was always coated with a white fur. Washings from it had the identical odor which one always detected on her breath.

The fetor ex lingua was not eliminated until we started the use of antibiotics (aureomycin). Cleansing of the tongue alone produced only temporary relief of the fetor.

Lately, in searching for foci of infection of the upper respiratory tract, I came across patients with fetor ex lingua. Upon eliminating the fetor, the remote malady for which a search for a focus was made, also began to resolve without any other infection presenting itself. I then made it a rule to examine the whole tongue routinely. When coating is present and washings have a characteristic odor of putrefaction, the aim is to clear up that condition first. Many times the patient got well without further treatment, and without finding another focus of infection. Crowe's[14] statement regarding "the theoretical explanation on which is based the otolaryngological treatment and preventive measures for recurring upper respiratory infections and their complications" can be applied to the tongue's acting as a focus of infection. Pyogenic bacteria grow on the coated tongue just as they would in a test tube. These cannot initiate infection; but when the tongue becomes infected with a virus, which is an intracellular parasite, it opens a pathway of infection. The infection may thus be carried through the blood stream and act as a focus of infection for remote diseases. Several cases of iritis cleared up by simple cleansing of the coated tongue with aureomycin in suspension (50 mg. to one dram of normal saline) and aureomycin internally, as exemplified by the following case:

Case 4: A. C., male age 20, came under my observation June, 1939. Diagnosis was iritis. Conservative treatment made no impression. Infected tonsillar tags were found and cauterized, and the eye cleared up quickly. May 7, 1946, he had a similar attack of iritis. Empyema of both antra was present and cleared up with washings. The eye cleared up very quickly with simple treatment of atropine. Dec. 3, 1950, he had another attack of iritis. This time no focus could be found. Milk injections were given with no appreciable effect. He had a fetor ex ore with the tongue coated and washings elicited a foul odor of putrefaction. This was cleaned and aureomycin given internally and the eye cleared up quickly.

Another case of iritis also of recurrent nature was given aureomycin but with very little effect. The tongue was found to be infected and when this was cleared up the eye got well.

One case of optic neuritis was treated with cortisone, aureomycin, and milk injections with no effect. She had complete physical and laboratory tests taken, including a spinal tap, with all negative findings. She had a fetor ex ore which was found actually to be fetor ex lingua. This was cleaned up with aureomycin suspension locally, and the clearing of the eye followed very quickly.

Two cases of dacryocystitis, as mentioned above, have been cleared up with the elimination of foci of infection on the tongue. One case was of a purulent nature which had to be incised externally, and the other had simple epiphora.

Ten cases with Ménière's syndrome were also cleared up by the hygiene of the tongue. They were all of a toxic nature, but no focus could be found other than the coating of the tongue with putrefactive material. These may be exemplified by the following case:

Case 5: A. D., male, age 60, was treated for catarrhal deafness with the Eustachian tube obstructed. He was doing fairly well when he began to have evidence of Ménière's syndrome. I diagnosed this case as a toxic labyrinthitis, but I could not find a focus of infection. He was given two courses of aureomycin, and he improved somewhat. When medication was stopped his symptoms recurred, and he became a chronic vertiginous patient. Upon the slightest provocation he would have vertigo of variable degrees and duration. Further checking on him elicited a coated tongue, washings from which emanated the characteristic odor of putrefaction. Treatment of the tongue was then instituted, the patient improved very rapidly.

Since then nine cases of Ménière's syndrome were seen in patients with more or less of a catarrhal deafness. The impairment of hearing was not affected by the attack nor was tinnitus complained of. They all had coated tongues with odor of putrefaction. When treated as outlined above, good results were obtained in all of them. Three patients came soon after the first attack. Their tongues were coated only a short while and responded to local treatment alone.

Conclusion: The coated tongue may act as a focus of infection and sometimes be the only focus for a varied number of diseases. Every coated tongue should have washings examined, particularly as to odor. If putrefaction is present, the tongue should be cleaned thoroughly with antibiotics and antibiotics given internally. When putrefaction has been existing for a long time, treatment should be prolonged, since recurrence of the odor and the malady are likely to reappear.

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interior of the nose. Here, attached to the septum, it presents as a grayish, semitranslucent, sessile tumor which is covered by septal mucosa.

Intranasal meningoceles have been confused with polypi. This error should not occur if one remembers that polypi in infants are exceedingly rare, that they are pedunculated and originate near the ostia of the sinuses on the lateral side of the nose.

Other criteria have been suggested to aid in the differential diagnosis. Compression of the jugular veins may produce a light reflex and distension of the meningocele. Lumbar puncture and drainage may decrease its size and tension. X-rays have demonstrated the defect in the cribriform plate.

The treatment of intranasal meningoceles has been far from successful. Only three cases reported in the literature have ended in recovery. Biopsy and aspiration have resulted in a fatal meningitis in many cases.

The treatment of choice is ligation of the pedicle either via the radical frontoethmoid approach or by craniotomy.

CASE REPORTS.

The first case is that of a three-year-old white male who has a symptomless, soft, compressible tumor mass at the root of the nose, 2 cm. in diameter.

The diagnosis of frontal meningocele was made, but since no immediate treatment was advised the patient was not seen again.

The second case is that of a white male infant, 23 months of age. At about nine months of age he developed a constant discharge and obstruction of the left nostril.

He had a nasal operation in January, 1950, an adenotonsillectomy in March, and another nasal operation in April.

On July 17, 1950, when first seen in consultation the patient had an obvious nasal obstruction on the left. The nostril was occluded by a pale, soft tumor mass covered by glary mucus. Adenoid tissue partially occluded the left choana.

Following a course of X-ray therapy, the nose was dry and the child breathed normally for several months.

When next seen, Nov. 27, 1950, the nasopharyngeal lymphoid tissue was absent, but his left nostril was still obstructed.

On the suspicion that we were dealing here with an intranasal meningocele, he was examined under general anesthesia. After cleansing the left nostril of mucus, the tumor was found to be continuous with the mucosa of the septum and free from any lateral attachment. The nostril was carefully packed with adrenalin gauze so as not to traumatize the tumor, and the packing left in place for five minutes. When it was removed the tumor had disappeared, but the sac could be seen filling the olfactory groove. It gradually expanded while under observation.

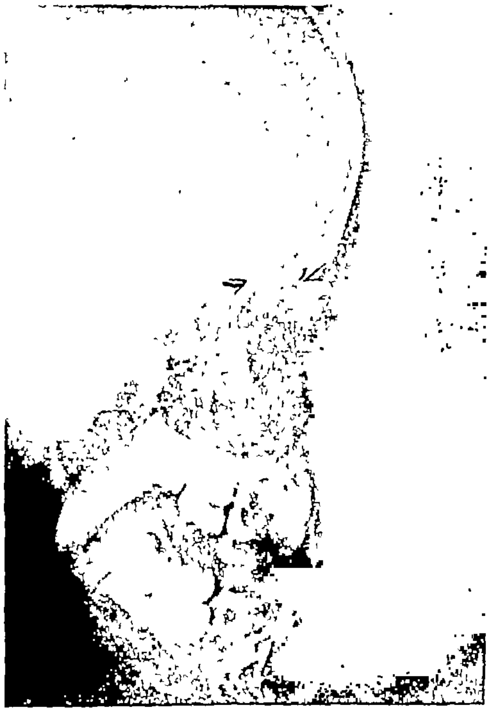

Fig. 1. The lateral X-ray of the anterior part of the skull revealed the dehiscence in the cribriform plate between the arrows.

On the basis of these findings, the diagnosis of intranasal meningocele appeared to be warranted. Accordingly, the patient was referred to Dr. William Lipscomb for craniotomy. Dr. Lipscomb's report follows:

The diagnosis of intranasal meningocele was made by Dr. Beyer and Dr. Blair, so when the patient was referred for treatment he was admitted to Children's Hospital where X-rays of the skull were taken which revealed, in a lateral projection (see Fig. 1), a dehiscence in the cribriform plate, anteriorly. In an anterior tangential X-ray view (see Fig. 2)

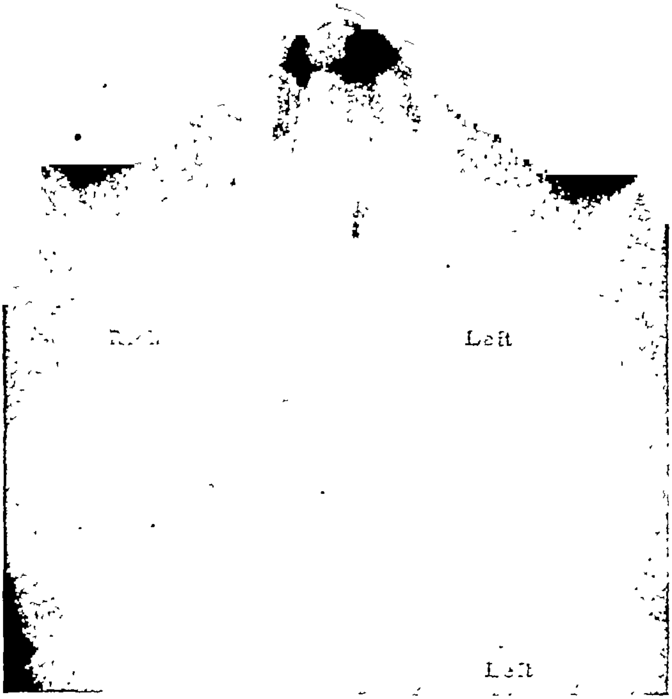

Fig. 2. The anterior tangential X-ray of the skull revealed the marked enlargement of the left naris.

of the skull the left naris was much larger than the right. On Nov 29, 1950, under general inhalation, intratracheal ether anesthesia combined with a field block of 1 per cent procaine containing 1:200,000 epinephrine, a left transfrontal craniotomy was done. An extradural approach was made toward the sphenoidal ridge where a long dural incision was made so that the left frontal lobe could be elevated and dislocated posteriorly. Near the anterior tip of the left frontal lobe on the medial, inferior aspect a small stalk (approximately 3 mm. in diameter) of arachnoid like tissue was encountered which was binding the arachnoid over the tip of the medial, inferior aspect of the left frontal lobe near the longitudinal fissure to a depression in the olfactory groove of the anterior *fossa*. The stalk was doubly ligated with No 50 cotton sutures and sectioned between the ligatures. With the bayonet forceps the dehiscence in the cribriform plate could then be easily palpated. It measured approximately 1 cm in length and 3 mm in its widest dimension. The dura was scarified for approximately 1.5 cm surrounding the sectioned stalk over the cribriform plate and a free pericranial graft was taken and placed to cover the sectioned end of the arachnoidal stalk and the bony dehiscence. The area was suctioned dry in order to promote adherence of the graft. The brain was then allowed to fall into normal position and the wound closed in layers. A Penrose drain was left in the wound two days and 10,000 units penicillin were injected into the wound after closure. In order to get the brain to tampon the pericranial graft over the bony and dural defect an effort was made to keep the patient lying on the right side with the head in right oblique position. In a 23 month-old child such an effort was only partially successful.

On Jan. 19, 1951 (51 days postoperative), the patient was brought to the office by the parents who reported he was "nervous and didn't sleep well, but that he had had no nasal symptoms." On examination, however, he was alert, agile, interested and happy. Neurological examination and an external examination of the nose revealed the little fellow to have no nasal drainage and to have no obstruction to the passage of air.

CONCLUSIONS.

1. Meningoceles occur very rarely.
2. Intranasal meningoceles may be confused with polypi.
3. Aspiration and biopsy are dangerous procedures.
4. X-ray studies proved to be a valuable diagnostic adjunct.
5. Intracranial ligation of the pedicle appears to be the treatment of choice.

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this department in one month, both under the diagnosis of pneumonia—one of them almost moribund at the time of admission:

Case 1: No. 107/50. Girl, three months old. Admitted Feb. 3, 1950, and discharged on March 20, 1950.

The child was born two to three months before term. Bottle-fed. One week before admission showed restlessness, dyspnea, vomiting and a few thin stools.

Physical Examination: On admission, the child was markedly exhausted, grayish, breathing very rapidly (respiration rate 70 per minute). Dilatation of the nostrils and pronounced inspiratory retraction of the suprasternal notch and epigastrium, but no stridor.

Auscultation of the Lungs: Semimoist, subcrepitant rales heard over the anterior and posterior surfaces. Temperature, 36.8° C.

Treatment: Sulfathiazole, penicillin, streptomycin and oxygen.

As atelectasis was suspected, an X-ray picture was taken of the child in the ward with a portable X-ray apparatus. It showed atelectasis of the right upper lobe (see Fig 1A). On the day after admission, bronchoscopy was performed with the Negus baby bronchoscope (Dr. Riskaer). A very abundant tenacious greenish-yellow secretion was found in the trachea and both main bronchi, especially in the right. The mucous membrane was swollen, again especially on the right side, where the lumen was distinctly narrowed. The secretion was aspirated (cultures gave growth of pneumococcus type 33 and B. coli), and oxygen was administered. After this, the respiration improved considerably.

Eight hours later the condition of the child again deteriorated, and death seemed imminent. The respiratory rate was about 100 per minute in spite of stimulation with lobeline and oxedrin.

Bronchoscopy was repeated, with aspiration of a considerable amount of tenacious secretion, though distinctly less than before. On introduction of the bronchoscope, which was easy enough, the respiration stopped, but was soon re-established by administration of oxygen through the bronchoscope. For the next several days the condition of the child improved strikingly. On discharge, the child was perfectly well. The Roentgenographic findings are reproduced in Fig. 1.

Case 2: No. 182/50. Girl, two months old, breast-fed. Admitted March 4, 1950, and discharged March 15, 1950. The child was born at term by spontaneous delivery. Previously well. Four days before admission she developed coryza with cough. The temperature did not exceed 37.5° C. On the day before admission the child was distinctly dyspneic, crying and restless. She was treated at home with steam and sulfathiazole—without any improvement.

Physical Examination: On admission the child was lively and not particularly distressed. Respiratory rate 40 per minute, with distinct inspiratory retraction of the epigastrium.

Auscultation of the Lungs: Breath sounds weak over the right posterior surface, and a few subcrepitant rales. Temperature 37.2° C. *Clinical Diagnosis:* Bronchopneumonia.

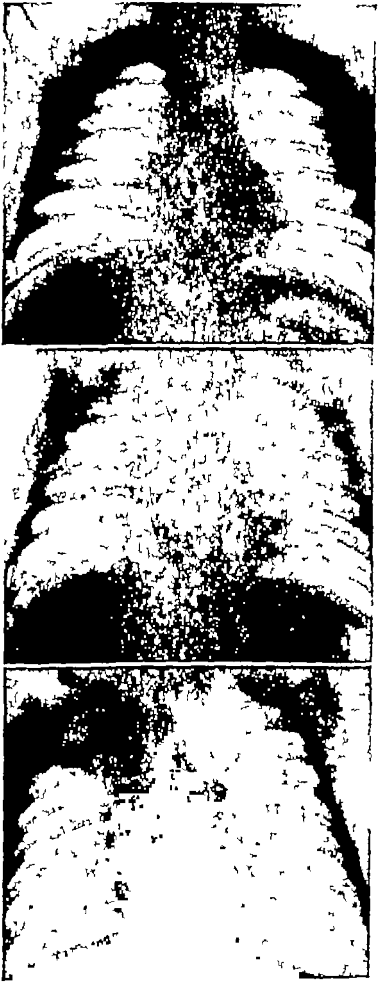

Fig 1-A

Fig. 1-B

Fig. 1-C

Fig 1 A, 1 B, 1 C. Case 1. Girl three months old. (A) Atelectasis of the right upper lobe (Feb. 4) (B) Three days after bronchoscopy with aspiration. Atelectasis almost subsided (Feb 7) (C) Atelectasis cured (Feb 11)

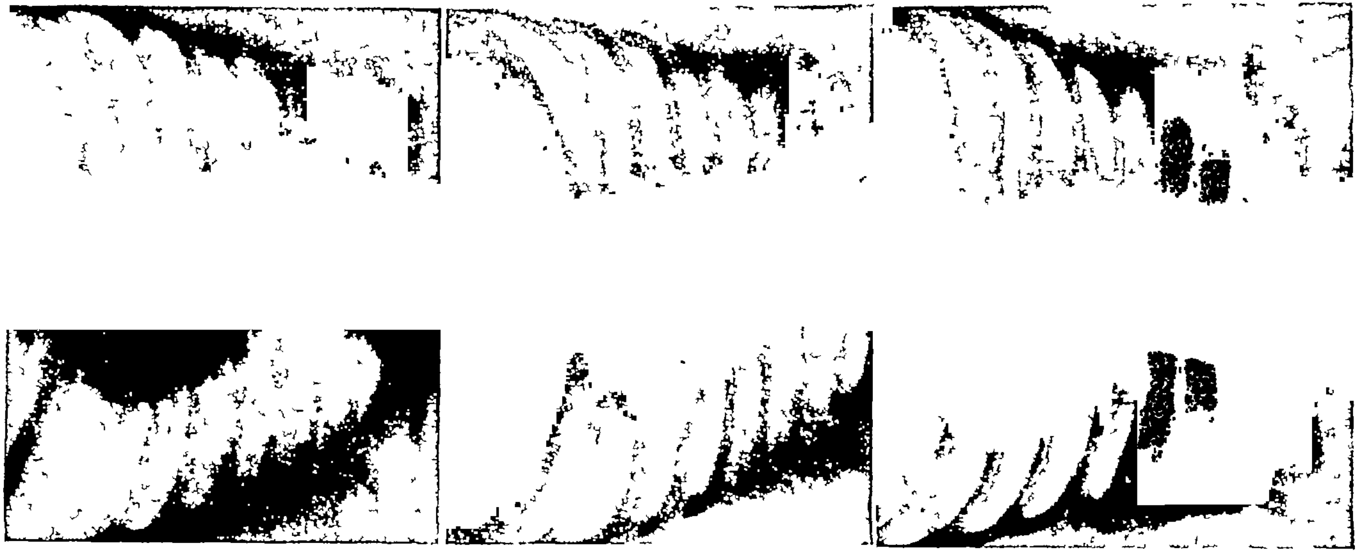

Fig 2-A

Fig 2-B

Fig 2-C

Fig 2-A, 2-B, 2-C Case 2 Girl, two months old (A) Atelectasis of the right upper lobe (March 5) (B) Twenty four hours after bronchoscopy and aspiration (March 6) (C) Three days after bronchoscopy with aspiration. Atelectasis cured (March 8)

Treatment Penicillin streptomycin steam and oxygen The following morning (March 5) there was an obvious deterioration in the condition of the child X ray examination of the lungs (performed in the ward) showed atelectasis of the right superior lobe (see Fig 3A) Bronchoscopy was then performed On aspiration fresh amounts of secretion appeared continuously into the bronchus, so that 10 to 12 cc in all were aspirated (see Fig 3) *The bronchoscopy was tolerated well Cultures from the* aspirated secretion yielded growth of Gram positive cocci (staphylococcus aureus and enterococci) and Gram positive rods

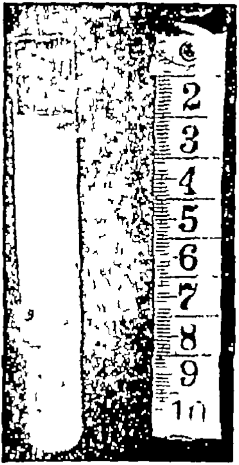

Fig 3 Case 2 Secretion withdrawn at the bronchoscopy

Five hours later the bronchoscopy was repeated and 3 to 4 cc. of very tenacious secretion were aspirated from the right main bronchus

After this the condition of the child improved considerably, and the respiratory rate fell from 80 to 52 per minute

Three hours later bronchoscopy was carried out for a third time, but there was only a scanty amount of secretion present.

Twelve hours later (March 6) the child appeared to be perfectly well and was discharged after 11 days in the hospital

The Roentgenographic findings are reproduced in Fig 2

The admission of these two patients within one month might be taken to indicate that *acute infectious atelectasis* is not altogether infrequent in infancy.

In order to look into the frequency of this lesion, therefore, we have gone through the autopsy records of those children who died under the diagnoses of pneumonia, bronchitis or laryngotracheobronchitis fulminans in the pediatric department of Sundby Hospital and Blegdam Hospital (Epidemic Diseases)[*] for the last five years.

The frequency of atelectasis is shown in Table 1.

TABLE 1.

Atelectasis demonstrated by autopsy in 178 children who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis in 1945-49.

	Sundby Hospital				Blegdam Hospital			
	+ Atelectasis				÷ Atelectasis			
Age	Focal	Extensive	Total	No Atelectasis	Focal	Extensive	Total	No Atelectasis
3 weeks-3 mons.	6	3	9	18	4	7	11	10
3- 6 mons.	2	4	6	2	10	4	14	18
6-12 mons	0	5	5	4	12	7	19	27
1- 2 yrs.	0	1	1	2	7	2	9	12
2-yrs.	0	0	0	2	1	1	2	7
Total	8	13	21[*] (43%)	28 (57%)	34	21	55[†] (43%)	74 (57%)

[*] In three of these cases the clinical diagnosis was laryngotracheobronchitis.
[†] In two of these cases the clinical diagnosis was laryngotracheobronchitis.

The figures given in Table 1 are the absolute minimal figures, as in a number of these cases no histological examination was made, and atelectasis cannot always be distinguished from pneumonia by macroscopic examination alone.

In this material the diagnosis of atelectasis was made clinically only in *one case* — that of a boy, three months old, who was admitted to Blegdam Hospital for whooping cough. The diagnosis was made Roentgenographically, and bronchoscopy was not performed. The child was treated with penicillin and sulfathiazole, but died nine days after admission—complete atelectasis of the right lung developed gradually (see Figs. 4A and 4B).

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[*] We are greatly obliged to Prof. H. C. A. Lassen, M.D., Chief of Blegdam Hospital, for permission to make use of the case records.

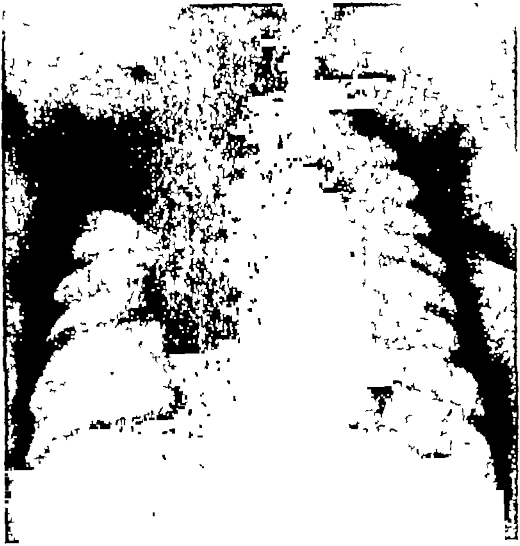

Fig 4-A. Boy, three months old Atelectasis of right upper lobe (Sept 26, 1948)

Among the 129 patients in the autopsy material from Blegdam Hospital, 1945-1949 (*cf.* Table 1), X-ray examination was performed only on two other patients (two boys, five and eight months old). In each of these cases the X-ray diagnosis was pneumonia, but autopsy (carried out one and two days after the X-ray) revealed the presence of atelectasis in both.

and no pneumonia. At the X-ray examination no lateral picture was taken, and perhaps this may explain the erroneous X-ray diagnosis.

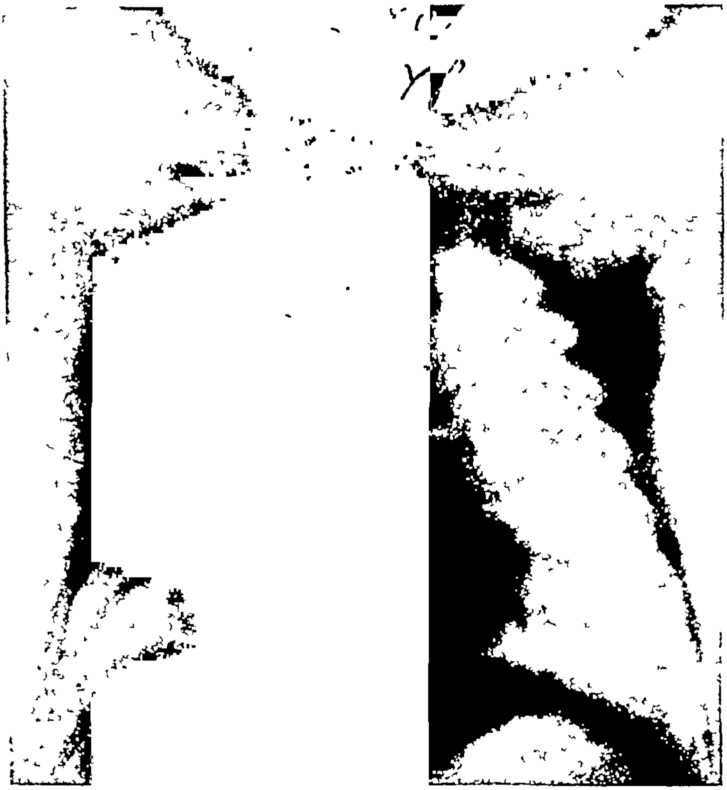

Fig 4-B Eight days later Total atelectasis of the right lung (verified on autopsy 24 hours later) Bronchoscopy not performed.

ETIOLOGY AND PATHOGENESIS OF ACUTE INFECTIOUS ATELECTASIS.

The term "atelectasis" means areas in the lungs where the alveoli contain no air but have undergone pronounced retraction and shrinkage. The prerequisite of this condition is active contraction of muscle tissue in the lungs, which thus distinguishes atelectasis from simple collapse of the lung (Xalabarder, 1949).

Clear-cut atelectasis is merely a stage of brief duration that is followed by vasodilatation with production of a serous, sometimes hemorrhagic, intra-alveolar exudate that may become infected from the bronchi. Atelectasis may be reversible at any of these stages. If it becomes more protracted, however, fibrosis and bronchiectasis may develop in the affected area.

In many cases the atelectasis process is preceded by bronchial obstruction; but it is the general opinion that it may also develop without obstruction, presumably through reflex action combined with hypoventilation (Xalabarder, 1949). Atelectasis due to obstruction occurs most often in infants. For instance, Anspach (1939) states that 80 per cent of such cases of atelectasis are found in children under two years of age, but he does not offer any explanation of this. Van Allen and Soo, on the other hand, discuss this question, pointing out that "collateral respiration" may prevent the development of atelectasis, even though small branches of the bronchi may become obstructed. In infants, however, the lungs contain relatively far more interstitial tissue than the lungs of older children and adults, resulting in a lesser possibility of "collateral respiration." This perhaps may explain the greater frequency of atelectasis in infants; furthermore, the risk of bronchial obstruction is also greater in infants, because of the small dimensions of the bronchi and the inability of infants to cough and expectorate.

SYMPTOMATOLOGY OF ACUTE INFECTIOUS ATELECTASIS CLINICAL.

The predominant feature in these children is severe dyspnea with pronounced retractions of the epigastrium and jugulum but no stridor. The chest is markedly distended, being almost fixed in the position of maximal inspiration, so that in spite of the great efforts of the thoracic wall, the breathing becomes very shallow. As a rule in such cases no rales are heard on auscultation. It is also a characteristic feature that the dyspnea does not satisfactorily improve with oxygen therapy—in contrast to pneumonia. The color of the face is grayish, associated with a state of shock. Definite cyanosis, therefore, is not a dominant symptom, appearing only occasionally on marked physical exertion, *e.g.,* when the child is crying.

Cough may be entirely absent, but in typical cases a so-called "frustrate" cough—*i.e.,* a dry, hacking, paroxysmal and ineffective cough—is present.

The temperature is often normal or only slightly elevated, as seen in children with severe pneumonia.

On auscultation—as in pneumonia—dullness, impaired respiration and rales may be demonstrated; but a sign of differential diagnostic significance is retraction of the affected side of the chest with displacement of the heart to the affected side, even though, as a rule, this may be ascertained only when the atelectasis involves the right lung and with diminished excursions of the chest wall and, sometimes the diaphragm, kept at a high level.

Bronchoscopy shows a very abundant amount of mucopurulent secretion in the bronchi.

ROENTGENOLOGICAL.

The X-ray picture is characterized by a shadow produced by the retracted atelectatic area, with a sharp convex border; the trachea and the heart are displaced to the affected side; the interlobar fissures are displaced, and the intercostal spaces diminished, sometimes with elevation of the diaphragm.

As these patients were markedly exhausted, the X-ray examination had to be performed in the ward. At first the Roentgenograms were failures, because the very rapid respiration (80 to 100 per minute) required a rather short time of exposure, and this was not practicable with the portable X-ray apparatus at our disposal. This difficulty was overcome, however, by means of a simple trick, *viz.,* by means of a pinprick the child was made to cry, and after a few vigorous screams the breathing stopped long enough for adequate Roentgenography.

DISCUSSION.

The clinical picture just described as characteristic of acute infectious atelectasis differs from that of the typical acute fulminating laryngotracheobronchitis primarily in the absence of any obstruction of the larynx, *i.e.*, dyspnea is the main symptom. On the other hand, it is well known that the occurrence of atelectasis may also play an important rôle in laryngotracheobronchitis—first described by Brennemann, Clifton, Frank and Holinger (1938) in America, and later by Boysen and Boysen (1942) and Arnesen, Bjerkelund and Boe (1947) in Norway, and by Söderling (1947) in Sweden. These authors also mention that in some cases of fulminating laryngotracheobronchitis the laryngeal obstruction may not be obvious or spontaneously may subside completely. In these cases a clinical picture also develops in which "pulmonary dyspnea" results from obstruction of the deeper air passages and possibly the formation of this atelectasis is the dominating feature; however, this clinical picture is probably a rather uncommon variant of fulminating laryngotracheobronchitis. None of these authors differentiate acute infectious atelectasis without stridor as a nosographic entity *per se,* which the clinician must keep in mind, especially in the diagnosis and treatment of pneumonia.

TREATMENT.

Aspiration of the secretion in the bronchi is the most important part of the treatment in that phase of acute infectious atelectasis where the dyspnea is most pronounced, and the patient is threatened with suffocation. As described in our case histories, the aspiration should be performed in connection with peroral bronchoscopy. This causes minimum upset and is easy to perform within a few minutes with the child in bed. It is tolerated well even by moribund infants. The lifesaving significance of this intervention was emphasized by its immediate effect on the condition of the patients and by the aspiration of the surprisingly large amounts of secretion. It is to be emphasized that bronchoscopy with aspiration should be looked upon as indicated whenever the clinical picture is suggestive of infectious atelectasis, even though the presence of this condition has not been verified Roentgenologically.

Tracheotomy, in our opinion, is contraindicated in children presenting this clinical picture, as no laryngeal obstruction was present and because such an operation would mean an added strain upon the child that might easily prove fatal to these exhausted children.

In contrast, both tracheotomy and aspiration would be indicated in typical cases of fulminating laryngotracheobronchitis—as emphasized by Jackson and Jackson (1936), Gittins (1932), and others who later discussed this disease. From their works it is evident that when tracheotomy is performed the aspiration is most often carried out in a "groping" fashion with a catheter inserted through the tracheal cannula. This technique must be considered as less effective than aspiration through a bronchoscope. This latter method is also advocated by Brennemann and collaborators whenever it is technically practicable, *i.e.*, when the instruments required and an experienced bronchoscopist are available.

In more serious cases of simple pseudocroup, tracheotomy will be sufficient in itself.

Besides bronchoscopy, the treatment of severely ill children also requires intensive therapy against the infection, a sufficient supply of fluid, the re-establishment of the electrolyte balance, and possibly blood transfusion in order to overcome the state of shock unquestionably present.

The combating of the infection should be started at once by treatment with antibiotics, etc., in order to ensure the best possible effect, bearing in mind that the nature and resistance of the pathogenic bacteria in the given case are unknown. Naturally, the aspiration should be carried out under sterile conditions, so that the proper bacteriological examination of the secretion and determination of the bacterial resistance may be carried out and further treatment based rationally on the results.

Our patients received both penicillin and streptomycin by injection and inhalation. The results of the cultures and the demonstration of the varying sensitiveness of the isolated bacteria to the antibiotics employed showed this combined therapy to be justified.

CONCLUSION.

1. Acute infectious atelectasis is not a rare condition in infants in connection with bronchitis and pneumonia. Autopsy on 178 infants who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis revealed atelectasis in 43 per cent of the cases.

2. The clinical picture of the lesion is so typical that the diagnosis can be made with a fairly high degree of probability, even on ordinary physical examination, but its verification requires X-ray examination with a portable apparatus on account of the exhausted condition of these children.

3. The most important therapeutic measure is early bronchoscopy and the aspiration of the secretion in the bronchi, and the immediate employment of antibiotics.

4. It seems rather likely that the relatively high death rate from pneumonia in infants despite treatment with penicillin and sulfa preparations is due to unrecognized and, therefore, untreated infectious atelectasis.

SUMMARY.

The development of acute infectious atelectasis is pointed out as a contributory cause of the strikingly high death rate from pneumonia in infancy. The records of two characteristic cases are reported. A brief review is given of the autopsy records for 178 children who died under the diagnosis of pneumonia, bronchitis or laryngotracheobronchitis, showing the presence of atelectasis in 43 per cent. The diagnosis and treatment are discussed. It is emphasized that administra-

tion of sulfa preparations and antibiotics is not sufficient, and that repeated bronchoscopy with aspiration is required in order to avoid suffocation in the most critical phase.

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GRANULOMATOUS ULCER OF THE FACE. A REPORT OF A CASE.*

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There have been extensive reviews of the literature and much discussion as to the etiology and cause of the granulomatous ulcers of the face and adjoining facial structures. Hoover[1] reviewed 60 cases of lethal granulomatous lesions involving facial tissues. Other cases have been reported by Levy,[2] Lierle,[3] Isbell[4] and others. Stewart[5] gave an excellent clinical review in his paper. Williams[6] gave a very lengthy discussion as to the possible causes and associated factors that may play an important part in these lesions. Recently he reported a case[7] that has been treated with hormones of the adrenal cortex. The following case is one in which the causative agent was isolated and the lesion was treated with the appropriate agent both locally and systemically.

A CASE REPORT.

A 74-year-old white female was admitted to the Massachusetts Eye and Ear Infirmary on April 27, 1950 with the complaint of swelling and redness around the lower lid of the right eye. She first noticed swelling on the right side of her nose about Jan. 1, 1950. The swelling and redness gradually increased and involved the right nasal bridge, the right infraorbital region and the cheek. There was no pain, no nasal discharge, no epistaxis, and no previous sinus disease except for a heavy feeling in the right antral region.

Examination revealed an indurated, swollen area over the entire right superior maxilla with marked tenderness over the right glabella region, ascending process of the right maxilla and lacrimal fossa. The infraorbital rim was intact. The buccogingival recess over the canine fossa was full and very tender to palpation. The rest of the physical examination was negative.

The past history revealed that the patient was in excellent health except for mild diabetes which was controlled by diet alone.

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*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

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Laboratory data on the first admission was essentially negative except that the X-rays showed some thinning of the right nasal bone and many abscessed teeth. The leucocyte count was 8,700, 59 per cent polymorphonuclear leucocytes.

On admission it was felt that this was possibly a malignant granuloma, but with the finding of many abscessed teeth, it was felt that these should be removed as a possible source of infection. The patient was put on a diabetic diet, 200,000 units of aqueous procaine penicillin and hot compresses to the area of involvement. The abscessed teeth were removed. While the patient was under the above treatment, a large slough developed in the area of the previous swelling. The bone underlying this area looked normal. It was felt that the patient could be followed satisfactorily as an outpatient and she was discharged, to be given daily injections of 300,000 units of procaine penicillin.

While being treated as an out-patient, it was noted that the area of ulceration was extending. A biopsy was taken and the report was returned as chronic inflammation. The patient was again admitted to the hospital and at this time the area of ulceration measured approximately 7 cm. in length by 1.5 cm. in width. There was undermining of the skin edges for approximately 4 mm. The base of the ulcer was covered with a small quantity of granulation tissue, and there was redness and induration extending beyond the ulcerated area (see Fig. 1). The lesion extended from the infraorbital region of the right eye, along

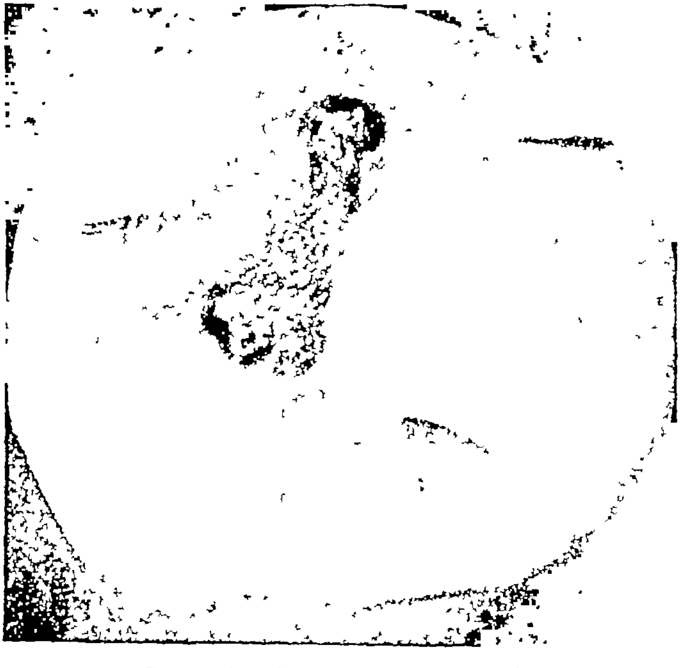

Fig 1. On admission to the hospital.

the ascending process of the right maxilla to the right nasal bone. It was approximately 3 mm. medial to the inner canthus of the eye.

The laboratory data on this admission revealed a leucocyte count of 8,700 with 67 per cent being polymorphonuclear. Blood sugar was 148 mgm. per 100 cc. of blood. The blood Hinton was negative. Cultures taken on admission and daily thereafter were planted on blood agar plates and meat broth tubes and were placed both aerobically and anaerobically. The initial culture revealed coagulase positive hemolytic staphylococcus auereus. This organism was found to be resistant to penicillin at 8 units per cc. and very sensitive to aureomycin and bacitracin. Following the report of the cultures and the sensitivity tests, the following plan of treatment was instituted:

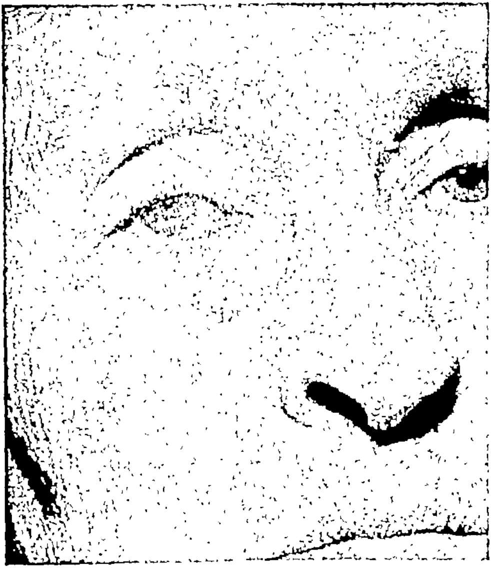

Fig. 2. Upon discharge from the hospital.

1. Cleansing of the ulcer with hydrogen peroxide to help remove all pus and debris. Thorough irrigation with saline to remove all traces of the peroxide and then 10 cc. of bacitracin containing 500 units of bacitracin per cc.

2. Sterile dressing with bacitracin ointment containing 500 units of bacitracin per gm.

3. Aureomycin 3 gm. daily and streptomycin 2 gm. daily.

Following this plan of treatment, on the fifth hospital day, granulations began to appear at the inferior lateral portion. After the sixth hospital day, all cultures were reported as "no growth." By the eleventh hospital day the epithelium had begun to grow from the nasal margin. On the fifteenth hospital day the streptomycin and the aureomycin were discontinued, but the local dressings were applied twice daily. The epithelium gradually closed over the area of the ulcer except for a small fistula that has remained in the region of the lacrimal sac. The patient was discharged from the hospital on the thirty-sixth hospital day. During her stay in the hospital, all urinalyses were negative for sugar. The patient has been followed at weekly intervals in the out-patient clinic and there has been complete healing of the area with minimal scar contraction. Repeated X-rays of the nasal sinuses fail to reveal any changes.

DISCUSSION.

A case of granulomatous ulcer of the face has been presented. The causative organism in this case was felt to be the staphylococcus aureus. This organism was found to be penicillin resistant, but senstive to aureomycin and bacitracin. It was treated with local dressings and oral aureomycin. It is felt that in infection such as this that repeated cultures should be taken and sensitivity results should be done to determine the appropriate agent to be used. In the present day of the great number of chemotherapeutic and antibiotic agents, along with the increasing number of organisms that are becoming resistant to these agents, there must be cooperation between the attending physician and the bacteriologist.

I acknowledge my indebtedness to Miss Anita Mangiaracine, bacteriologist at the Massachusetts Eye and Ear Infirmary, for her valuable bacteriological studies and suggestions as to treatment.

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A CASE OF CHOLESTEATOMA OF THE MIDDLE EAR IN A FIVE-MONTH-OLD INFANT.*

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Detroit, Mich.

Although the acute surgical mastoid has become a thing of the past, the problem of the cholesteatoma of the middle ear still remains to torment the otologist. The etiology, pathology and physiology, as explained by Wittmaak,[12] have been generally accepted by the majority of practicing otolaryngologists; however, periodically there appears in the literature a dissenting view.[1,8,9,10] The problem resolves itself to the basic premise: which comes first, the cholesteatoma or the chronic otitis media and mastoiditis? Some men have gone so far as to diagnose every case of chronic otitis media as a type of cholesteatoma.[8]

The rarity of this condition in infancy and childhood and the finding of a cholesteatoma only in the middle ear that has "run" for years make the hypothesis of the infectious etiology of cholesteatoma very plausible;[2,4-6] however, the occasional occurrence of cholesteatoma in an ear that has discharged for only a short period of time and the difficulty in explaining marginal perforations give strength to that group who believe the cholesteatoma to be the primary condition.[10]

When one reviews the history of the early description of cholesteatoma, he is surprised that the pathology and etiology are based on rather casual observations.[11] Wittmaak[12] considered the process of cholesteatoma formation as an attempted repair by nature. The squamous lining of the external canal grows into the middle ear to replace the

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degenerating mucous membrane. The formation of cholesteatoma will occur only if there is an inadequate outlet for drainage of the exfoliating material. Thus the attic and antrum, because they have a relatively small outlet, are ideally located for this formation. His views have been generally accepted, although he carried on no conclusive experiments to prove them.

Tumarkin,[3] on the other hand, feels that the cholesteatoma arises from the normal inflammatory reaction of the pavement epithelium of the epitympanum, retrotympanum and pretympanum. He believes that the pavement epithelium, which normally lines these areas, reacts to infection (and possibly other irritants) by proliferation and the throwing off of paper-like squamas. If the epitympanum, retrotympanum and pretympanum react as described, the above explanation of cholesteatoma becomes very plausible.

Kopetsky[4] has made a very practical division of cholesteatoma into the true and pseudo types. The pseudocholesteatoma is further divided into the primary and secondary. The primary type is associated with a dry ear and is believed to be due to retraction of the drum and then perforation; the secondary type is associated with a draining ear. It is generally known that a condition of true cholesteatoma can occur anywhere in the skull and is rarely (except when the temporal bone is involved) a direct concern of the otologist. Perhaps in the future the primary pseudocholesteatoma will be explained on the basis of Tumarkin's theory.

Recently at the Children's Hospital of Michigan a case of cholesteatoma in a five-month-old infant was observed. The case report follows:

Present Illness: D. T., a five-month-old white male child, was apparently perfectly well until one week prior to admission, Feb. 26, 1950, when he developed a cold with cough, running nose and irritability. The mother was not concerned until the morning of admission when she first noticed an asymmetry of the face. Patient remained irritable and the left ear began to drain. The mother denied any previous trouble, any postauricular swelling or any pulling at the ears. The child had been a normally developing child until onset of the present illness.

Past History: Essentially negative.

Birth and Development: Normal.

Family History: Essentially negative.

Physical Examination: Patient was a five-month-old, well-developed and well-nourished white male child. He was irritable but was in no acute distress and appeared only moderately ill. Hydration was good. Fontanelle was patent but not full. *Eyes:* Left eyelid was paralyzed, and patient could not close the eye. Right eye was normal. Pupils normal. *Ears:* Right drum was mildly injected. In the left external canal some yellow, creamy discharge was seen which obscured the drum. There was questionable mastoid tenderness on the left. The left-sided complete facial paralysis was the only other finding of importance.

Impression: Left acute otitis media and purulent mastoiditis with left facial paralysis.

Laboratory Data: Spinal tap was negative. Blood count showed marked leucocytosis with 29,500 white blood cells of which 51 per cent were neutrophiles. Culture of ear discharge revealed D. pneumonia.

Hospital Course: On the day of admission the left canal was aspirated. Some grayish green tissue was obtained, along with the purulent discharge and was sent to the pathological laboratory. The left eardrum was then visualized for the first time. There was seen a posterior inferior perforation extending superiorly to the membrana flaccida. The malleus

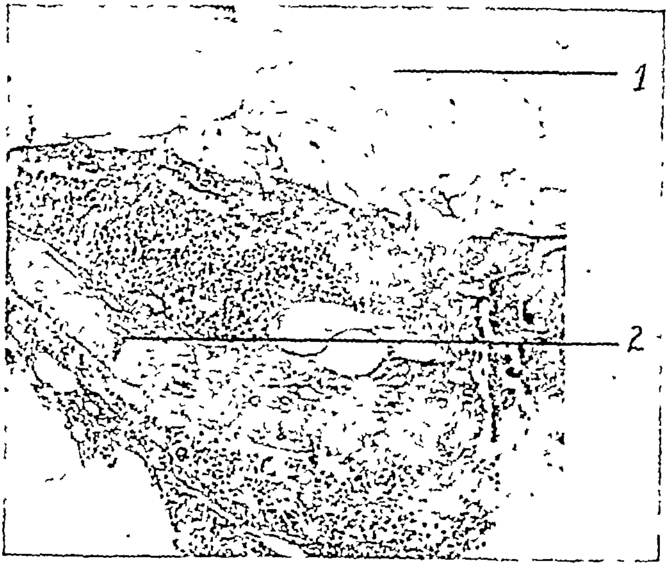

Fig. 1. Cross-section of tissue removed from external ear canal. ×150. (1) Epithelial detritus and keratin sheets in cyst contents. (2) Epithelial wall lining destroyed and desquamated; replaced by connective tissue and leucocytes.

appeared to be intact. The anterior drum was injected Through the perforation some of this grayish material was seen in the middle ear The external canal appeared normal The following day similar material was again present, again removed and sent to pathology. The drum had the same appearance. On March 8 1950 a pathological report of cholesteatoma was made on this tissue (see Fig 1) The facial paralysis persisted X-rays were taken but were of no diagnostic significance[7] On March 8, 1950, patient was taken to the operating room and under local anesthesia a left cortical (simple) mastoidectomy was done The bone was sugary in consistency, and in the antrum in addition to much pus, more of this grayish green tissue was found, sent to the pathological laboratory and again a diagnosis of cholesteatoma was made (see Fig 2) Drains were placed in the mastoid cavity and in the external ear canal The incision was not closed The patient left the operating room in fairly good condition *The postoperative course was complicated by a very severe diarrhea,* of questionable etiology but patient finally recovered Postoperatively he received penicillin and streptomycin and at the height of his diarrhea, he was started on aureomycin An adhesive tape splint was used on the left side of the face, and patient was given physiotherapy On the twenty seventh postoperative day he was discharged The postauricular incision had healed; the left eardrum was intact, and there was questionable improvement of the left facial paralysis

He was followed in the out patient department, and his mother was to give him physiotherapy at home On May 17, 1950, it was noted that the facial paralysis was no longer present, face moved symmetrically The eardrums were examined and both appeared normal.

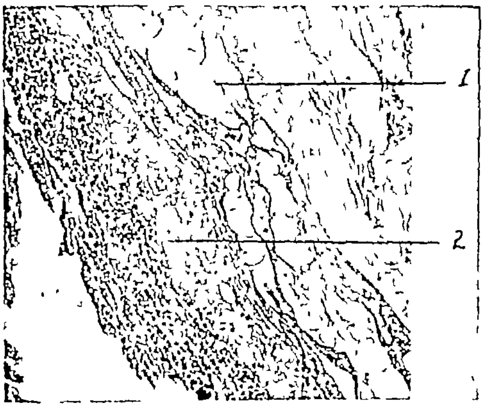

Fig. 2. Cross-section of tissue removed from mastoid cavity ×160 (1) Epithelial detritus and keratin sheets in cyst contents (2) Epithelial wall lining partially destroyed and replaced by connective tissue and leucocytes

On June 14, 1950, patient returned for a routine postoperative visit. At that time the mother stated that the child had developed a cold with cough and nasal discharge one week previously. Two days prior to the visit, there was noticed a purulent discharge coming from the right ear and one day previously it was noticed that the right side of the face was paralyzed. On aspirating the right canal, there was seen a central perforation with a pulsating, purulent discharge. Ear culture revealed hemolytic staphylococcus aureus. There was questionable right mastoid tenderness and a complete right-sided facial paralysis. The left ear was normal. The left face was normal. The patient was admitted, and on June 17, 1950, a right cortical (simple) mastoidectomy was done. The findings were those of an acute mastoiditis. No cholesteatomatous material was found. Patient was placed on penicillin and sulfadiazine. The postoperative course was uneventful, and on July 1, 1950 (20 days postoperatively), patient was discharged home. At that time his postauricular incision had healed, both drums were intact, and the facial paralysis on the right was minimal.

On July 28, 1950, patient was seen in the out-patient department. At that time there was no facial paralysis, the right and left drums were normal, and patient was doing very well.

COMMENTS.

This case, in itself, cannot solve the problem of cholesteatoma of the middle ear; however, the occurrence of a cholesteatoma in a five-month-old child without previously known ear trouble[1] is worthy of note. We realize that Hemsath[3] reports changes in the middle ears of newborns and embryos, due to foreign body reaction and infection.

It is the author's opinion that the work of Tumarkin[8] concerning the etiology of cholesteatoma deserves careful study. The evidence he presents is certainly logical, although his case presentations are not too conclusive.

This case proves that one cannot rule out cholesteatoma because of the duration of the aural discharge or the age of the patient. The occurrence of the facial paralysis, first on one side and then the other, is certainly an interesting phenomenon—especially since the paralysis was only temporary.

SUMMARY.

A case of cholesteatoma of the middle ear in a five-month-old infant is presented. The problem of etiology and pathology is discussed.

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CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Dunlap Hotel in Jacksonville, Ill., on Sept. 21, 22 and 23. The guest speakers will be Dr. Theo. E. Walsh, of St. Louis; Dr. James H. Allen, of New Orleans, and Mr. Henry C. Black, Professional Management, of Detroit.

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The Congrès Francais d'Oto-Rhino-Laryngologie will be held in the Grand Amphithéatre of the Faculté of Médicine, Paris, Oct. 22, 23, 24 and 25, 1951, under the direction of the presiding Dr. M. Bouchet, of Paris, and Honorary President de M. le Prof. Léon Binet, member of the Institute.

For further information, address Dr. H. Flurin, Secrétary Général — 19 Avenue MacMahon, 17e, or Dr. H. Guillon, Assistant Sécretary Général, 6 Avenue MacMahon, Paris, 17e.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

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Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.

Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

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Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico; Maico Model J.

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Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

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Rochester Model R-1; Rochester Model R-2.

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(See Micronic.)

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Manufacturer Sonotone Corp, Elmsford, N Y

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RHINOLOGY IN CHILDREN; RESUME OF AND COMMENTS ON THE LITERATURE FOR 1950.

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In this resumé of the literature pertaining to rhinology in children for 1950, the same journals have been covered as in previous reviews plus four major pediatric journals. The same order of discussion has been followed as before. The brevity of some of the sections makes obvious the slackness of those specialists who have the knowledge and facilities to write on these subjects

GENERAL ARTICLES ON RHINOLOGY.

As chairman of the Section of Internal Medicine at the 1950 Annual Meeting of the American Medical Association, Bloomfield[1] contributed an article on some problems of the common cold which should be read in its entirety. His conclusions were as follows

"The common cold is a specific virus infection which may be thwarted by resistance of the host or promoted under some conditions by exposure to cold or by other influences. Infection may come from without, from a patient or perhaps from a carrier, and carriers may catch cold from autoinfection. There is no solid evidence that the true cold is influenced by

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antihistaminics or antibiotics, nor is there any reliable method of prevention. Treatment remains symptomatic — rest, safeguarding, local applications and general sedatives. The true cold is often confused with the various forms of traumatic rhinitis, which are not the common cold at all and which may be amenable to various forms of suitable treatment, such as antihistaminic or antibiotic therapy. Secondary complications caused by ordinary bacteria are likely to occur during the course of the common cold. These may be helped by the use of antibiotics."

The author added a postscript that since the paper was written, several carefully controlled studies of the effects of antihistaminics in colds have appeared. In some no difference could be detected between the effects of the drug and a placebo. Feller and his associates found no alteration in the course of experimental colds produced in volunteers by virus instillation. In two other studies the observers felt that the early nasal symptoms were helped by antihistaminics but did not feel that the course of the ailment was otherwise altered.

The *British Medical Journal*[2] discusses claims made by a number of observers that antihistamine drugs may abort or beneficially modify the course of the common cold. It is of the opinion that in the most favorable circumstances it seems inappropriate to use the word "cure" in connection with the effect of antihistamine drugs on the common cold in the same sense in which it can be used, for example, with reference to penicillin in staphylococcal infection; nevertheless, even if symptomatic relief is afforded and the course of the disease modified, such treatment may be of some value. It is clear that further controlled experiments should be conducted before the antihistamine drugs are accepted as a routine form of treatment in this condition, and the Medical Research Council has agreed to arrange an investigation. Among the more obvious reasons for caution is the fact that, in addition to the mild sedative and gastrointestinal side-effects so commonly experienced after taking these substances, a number of serious and unusual reactions have now been recorded. These may indirectly endanger life, a hazard that is not usually related to the common cold itself. What appears to have been the first death in Great Britain after an overdose of an antihistamine drug was reported by Tobias in the *British Medical Journal* in 1949. The advertising of antihistamine drugs as "cold-cures" in the United States is a matter of concern to the Food and Drug Administration, which, it is reported, is ready to take court action if it could be proved that the drugs had caused injury.

The Department of Preventive Medicine, Western Reserve University,[3] closely studied family groups and volunteers for inoculation with the virus of the common cold. Their conclusion was that in neither group could the antihistamine drugs be shown to have any value in the prophylaxis or treatment of respiratory illness.

Fabricant[4] states that sufficient time has elapsed and enough convincing evidence has been accumulated at leading medical centers in the United States and Great Britain to establish the fact that despite the extravagant and irresponsible claims made, the common cold cannot be abolished by antihistaminic drugs.

The notion that allergy is mainly responsible for the common cold has never been substantiated by reliable investigators.

That antihistaminic drugs are not innocuous is becoming progressively evident. Instances have been reported of users of these agents falling asleep while at work, while operating machinery or while driving automobiles. Children have died after the accidental ingestion of large doses of antihistaminic drugs.

The failure of the antihistaminic drugs in the treatment of the common cold has been demonstrated in a series of carefully controlled studies in the United States and Great Britain. In Great Britain, controlled studies conducted under the auspices of the Medical Research Council at the request of the Ministry of Health provided convincing evidence of the worthlessness of antihistaminic drugs in the prevention and treatment of the common cold. In the United States, reliable controlled studies have been conducted by various investigators working independently at Cornell University, the Western Reserve University School of Medicine, the University of Minnesota, the University of Illinois College of Medicine, the Chicago College of Optometry, the United States Naval Training Station at Great Lakes, Ill., and the United States Military Academy at West Point.

A correspondent[5] asked the *Journal of the American Medical Association* for the opinion of pediatricians with regard to treating acute head colds in infants and was answered as follows: "Many physicians advise the use of nose drops which contrict the blood vessels of the mucous membrane. Many mothers request the use of nasal medicaments to relieve discharge and obstruction; however, there is increasing opposition by physicians and rhinolaryngologists to the use of these remedies. They have expressed the opinion that 'nose drops' constrict the vessels, subepithelial capillaries and arterioles and also the venous sinuses. If vasoconstriction is protracted, vasodilatation results; sometimes the shrinking and dilatation of the nasal mucosa may lengthen the infective process and increase the danger of sinus involvement or ear infections. If ephedrine or other vasoconstrictor drugs are prescribed, not much should be expected of their curative value; they should be used in dilute form, at long intervals between dosage and only for a short time. In acute cases they are of doubtful value, though they may be tried in the presence of nasal obstruction and severe discomfort and restlessness. In subacute sinusitis they may be of value by temporarily shrinking the mucosa of the nasal cavities, enlarging the drainage space and permitting escape of fluid from the sinuses."

Steinberg[6] reviews the literature concerning antireticular cytotoxic serum. This serum for use in man is produced by intravenous injection into animals of a saline extract of spleen and bone marrow taken after death from persons who had died from injury and who had had no acute or chronic disease. In 1942, reports from the Academy of Sciences of the Ukranian Socialist Soviet Republic had presented this serum as a new agent for clinical use in many pathologic processes on the basis of more than 2,500 clinical observations on such conditions as tonsillitis

In the ear, nose and throat service of the out-patient department of two hospitals in Philadelphia, Steinberg made observations on 17 boys and 10 girls over a period of one year Two children presented no definite problem and served as controls, 11 of those with various otolaryngologic conditions received one series of injections and 16 patients received two series of injections

Ten patients who received only one series of injections experienced no objective or subjective change in their symptomatology One child who received only one series derived greatest benefit observed from this form of therapy

Eleven patients who received two series of injections experienced no subjective or objective change. Four patients thought they had improved, but no changes were seen on physical examination One patient showed slight improvement and one, moderate improvement both subjectively and objectively

A table giving the data concerning the patients, and six illustrative case reports, are provided

From the results obtained and from a review of the literature, the author concludes that there does not seem to be a definite indication for antireticular cytotoxic serum in pediatric otolaryngology

SINUSITIS.

Fifty symptoms of chronic sinusitis in children are enumerated and discussed by Brown[7] These symptoms cover about every possible complaint in the upper respiratory area, however, his article does emphasize the importance of sinusitis in children, and he shows it occurs frequently, which is quite true The relationship of sinusitis and chest disease is stressed, and this is most important if early bronchiectasis and bronchitis in children are to be arrested before the lung changes become irreversible The author does not concede

In his second article Garrod[12] states that the indication for using penicillin is not a disease but an infection, wherever situated, by a sensitive micro-organism. So that treatment may be rational, a specimen for culture should be obtained before treatment is begun; a short period of chemotherapy can hopelessly obscure the picture. It is necessary to know the concentration necessary to combat the organism. Whether treatment will be of any value depends on how high a concentration can be obtained at the site of infection.

When streptomycin[13] is given by mouth its behavior is entirely different from that of penicillin; it is not destroyed, but neither is it absorbed; hence, this route is indicated only in order to produce an effect in the intestinal tract itself. To obtain a systemic effect, streptomycin, like penicillin, must be given by intramuscular injection. It reaches all important areas in the body except the cerebrospinal fluid; like penicillin, it has to be injected intrathecally for the treatment of meningitis.

Dosage varies within much narrower limits than that of penicillin, because streptomycin is by no means free from toxicity.

Streptomycin is actively bactericidal. There is no optimum concentration for this effect as with penicillin; the higher the concentration the more rapidly are bacteria killed. Its disadvantage is that bacteria can acquire a high degree of resistance to it with extraordinary rapidity. This is of such a degree that further treatment is futile; the change is also permanent. A sensible conclusion from these facts would seem to be that treatment should be brief and vigorous, allowing the least possible opportunity for bacterial recovery.

The only serious common toxic effect is on the VIIIth nerve, vestibular disturbances (vertigo and incoordination) being produced first, and deafness in more severe cases.

The main clinical indications for streptomycin are tuberculosis, various infections due to Gram-negative bacilli and occasionally coccal infections if they are abnormally penicillin-resistant.

Aureomycin and chloromycetin[14] are cautiously reviewed in Garrod's fourth article because when it was written comparatively little data on them had been determined in Great Britain.

Whereas penicillin and streptomycin are both bactericidal, aureomycin and chloromycetin are purely bacteriostatic in their action. Habituation, an ever-present threat to the usefulness of streptomycin, is not likely to impair either of these drugs, whether in the treatment of individual cases or by the general propagation of resistant strains.

These two antibiotics have a wide range of activity, overlapping that of penicillin and other chemotherapeutic agents to a considerable extent. It is by no means all-embracing. Neither drug has any useful action on the tubercle bacillus. Generally speaking, chloromycetin is the more effective against Gram-negative species and aureomycin against Gram-positive. Neither of the new antibiotics has much scope as a local application.

Studies in the use of aureomycin in children are presented by Whitlock, Hunt and Tashman.[15] They found that therapeutic blood levels for children resulted from a dosage of 11 mgm. per kilogram of body weight when the aureomycin was given by mouth at four hourly intervals. This was found to be the maximum dosage which was tolerated without giving gastrointestinal symptoms. The bitter taste makes its administration to small children difficult, and they found chocolate milk, chocolate syrup and lemon syrup the most satisfactory vehicles for overcoming this factor.

Aureomycin may be given intravenously to children, when necessary, in doses of 6.6 mgm. per kilogram of body weight every 12 hours. The drug should be injected slowly, taking two or three minutes to complete the administration.

Intramuscular and rectal administrations are not advised. Until further studies are completed on the photosensitivity induced by aureomycin ingestion, avoidance of exposure to the sun is suggested while the patient is taking the drug.

ALLERGY.

Walsh[16] states that perusal of 87 recent articles on vasomotor rhinitis leaves the reader with three impressions, that *a.* vasomotor rhinitis is due to a hypersensitivity to some foreign protein, be it inhalant or food; *b.* antihistaminics will relieve its symptoms; and *c.* specific desensitization or dust- or histamine-therapy will cure the condition. His experience contradicts these impressions.

Vasomotor rhinitis should be thought of not as a disease of the nose but as a manifestation of a systemic disease, the result of which is a sympathetic-parasympathetic imbalance resulting in peripheral vasodilatation, edema and secretion.

He considers that four factors lead to such an imbalance: *1.* allergy, *2.* endocrine dysfunction, *3.* dietary insufficiency, *4.* psychosomatic factors, and *5.* a combination of any of these.

He mentions certain cases illustrative of endocrine dysfunction resulting in vasomotor rhinitis. In his experience where a low basal metabolic rate is combined with a high blood cholesterol, the results from thyroid therapy are good; with a low metabolic rate and a normal blood cholesterol there are fewer good results. In these cases it is important to give sufficient thyroid to be of value. One of the patients improved by thyroid therapy was a boy 12 years of age. Walsh also claims that the nose reacts to abnormal function of the ovaries and of the testes and cites illustrative cases.

He deprecates the current fashion of considering a patient with a chronically stuffy nose, postnasal drip, frequent colds, "sinus" and so on as suffering from a true protein hypersensitivity to the neglect of the factors he has mentioned. He also quarrels with the too easy acceptance of eosinophilia as a diagnostic finding in allergy to the exclusion of other possibilities. Eosinophiles are present in secretions and tissues in allergic rhinitis, but their presence is not necessarily diagnostic of allergy; they may be present in other conditions.

In the course of an article on diagnostic problems in the study of asthma in children, Ratner[17] describes the differential diagnostic procedure of the study of the nasal secretions. It is simple and direct, and it vividly delineates the extent and character of the cells present The child blows his nose onto waxed paper. The collected secretion is spread on a slide and stained with Hansel's special stain, to be viewed under the microscope No special counting is necessary, but a panoramic view of the slide discloses whether eosinophiles or neutrophiles predominate, or whether the secretion is mucoid and contains preponderantly epithelial cells, bacteria or foreign material One can then state that the nasal condition is of allergic, infectious or irritative origin Whether this is a foolproof method he is not prepared to state, but for sheer objectivity it is far in advance of any method previously available, and he would not do without it.

CONGENITAL ABNORMALITIES

McGovern[18] *considers that the development and physiology* of the nose and paranasal sinuses is apparently little affected by total unilateral and bilateral choanal occlusion Many reports have been made and X-ray studies have been exhibited to illustrate complete development of the sinuses in cases of choanal atresia. Biopsy studies of the nasal mucosa have shown no departure from the normal structure in the cases here reported, and in other recorded cases The physiological activity of the nasal cilia appears to be unaltered by the presence of this congenital anomaly These findings suggest, as Boies has stated in the discussion of Wilkinson's paper, "that nasal respiration is not important to the development of the sinuses" nor, it would seem, to the normal physiology of the nose.

Sinusitis is an uncommon rather than a common complication of congenital choanal atresia These patients are not prone to acute infections of the nose and sinuses, the incidence of acute rhinitis is less than average. The history of continuous "colds" is a result of the periodic drainage of the retained secretions

The finding of aural disease and hearing impairment associated *with choanal atresia is not common Middle ear dis*ease, if present, is often found on the unobstructed side. The audiogram is usually reported as normal. The tympanic membranes are usually described as dull, lusterless, thickened or retracted. The interesting audiogram in Case 1 shows a bilateral perceptive type of impaired hearing. Fig. 8 illustrates an essentially normal audiogram of a 45-year-old colored woman (Case 3) with total right posterior nares occlusion. Wilkinson reported improvement in hearing in one case after surgical correction of the occlusion.

Adult patients with unilateral or bilateral choanal occlusion, as a rule, complain of little discomfort or distress because of their disability; surgical correction is often refused. The accumulation of the thick mucus is removed by the patient by gravity, the patient lowering the head and permitting the retained material to drain, assisted by pressure on the ala of the nose.

Bilateral choanal atresia in infants is of grave importance and requires prompt surgical correction. Failure to recognize this condition has resulted in the death of infants within the first week of life, under the mistaken diagnosis of asphyxia neonatorum. Thompson states that "the nose is the natural and instinctive channel for carrying on respiration. With rare exceptions all infants breathe entirely through the nose. If the nose of a sleeping child is gently closed, he will continue to make increasingly violent efforts to draw through it; when this proves useless, he will waken and gasp for breath, instead of simply opening and using the mouth as an airway. Even when mouth breathing is partially adopted to supplement the nasal airway, the latter remains the route preferred by instinct, and as much air as possible is drawn through it. Mouth breathing is an acquired habit." The diagnosis is suspected if the infant is unable to nurse, or has spells of suffocation and cyanosis which are relieved by crying and aggravated when nursing or sleeping. If the mouth breathing habit is established, the infant usually proceeds to normal growth and development except for the nasal obstruction and associated abnormalities. Unilateral atresia in the infant is largely a feeding problem and the prevention of malnutrition. Surgical correction may be undertaken in later life.

He concludes that congenital choanal atresia is an unusual but not rare rhinological anomaly The finding of these same defects in a mother and daughter suggests a definite hereditary tendency Complete bilateral or unilateral atresia has *surprisingly little effect on the development of the paranasal* sinuses or on the physiology of the nose or ears The association of sinusitis and choanal occlusion is uncommon Aural *disease and impaired hearing is infrequently* seen in choanal occlusion

Holmes[19] describes congenital triple nares in a female infant. This otherwise healthy baby had three hemangiomas, an anophthalmic right eye and a left nostril with two openings *The upper opening ended blindly under the nasal bones.* The lower opening was about the same size as the upper and extended inward and slightly upward to about the level of the pyriform aperture, where it narrowed before entering the nasal cavity The problem resolved itself into one of cosmetic improvement only, but, like the improvement of the other congenital deformities about the face, quite essential to the adjustment of the person to society He describes and illustrates his operative technique and by photograph shows the pre- and postoperative appearance

TONSILS

In the Semon lecture, Wright[20] reviews the available evidence as to the function of the subepithelial lymphatic system, *i e*, those collections of lymphoid tissue, namely, the tonsils, adenoids, Peyer's patches, appendix, which lie scattered through the length of the intestinal and upper respiratory tracts It would be generally agreed that the purpose of these structures is still ill-defined, they probably serve a common function as a specialized portion of the lymphatic system

A general description of the lymphatic system follows· it is a circulatory system acting in some specialized manner as an auxiliary to the blood circulation in the maintenance of the life of the tissue cell In man, it serves to return to the blood slowly the circulating material, particularly proteins, which cannot be readily absorbed by the blood capillaries The lymph nodes, interposed in the circulation, act as settling chambers and filters, removing inert foreign particles and micro-organisms from the lymph with a high degree of efficiency.

The reaction of the lymphatic tissue to injury or infection is especially noteworthy. The red streaks of lymphangitis and the tender swollen glands of lymphadenitis are visible indications of cell activity in the lymphatic tissue as a protective reaction to the infection. This reaction minimizes or prevents extension of the infection to the blood stream.

The specialized or subepithelial portions of the system are all so arranged as to bring their surfaces into intimate contact with the air and intestinal food streams and are particularly concentrated in areas where micro-organisms are most abundantly found. These structures reach their highest degree of development during early life and tend to retrogress in old age. They are known to permit the passage of organisms to the regional lymph glands and a most characteristic observation is that they bear the brunt of bacterial attacks in throat and intestinal infections.

Early life, the period of their greatest activity, is also the period in life when immunity is being acquired. The tonsils, and other subepithelial lymph structures, tend to increase in amount when subject to infection; and when some of these structures are removed, there is a definite tendency to enlargement of what remains, at any rate in early life. Thus it is obvious that the subepithelial lymph system has as its primary function the sampling of the micro-organisms in our environment and, as a result of this, defensive materials are elaborated with a resulting development of immunity.

That the development of the lymphoid tissue is stimulated by the presence of micro-organisms is supported by experiments on animals brought up under sterile conditions. These animals showed a subnormal development of lymphoid tissue.

From these observations the author postulates that the subepithelial portion of the lymphatic system was designed to permit the entry of limited quantities of organisms as "samples" which would stimulate the formation of immunity to future invasions.

The author theorizes that man's present lymphatic system was probably quite sufficient in the cave man era when there was little interchange between communities. Now, however, living in crowds in heated atmospheres, this mechanism for immunity is ill-suited to produce the same result; therefore, not infrequently, these structures themselves become diseased. He feels that this state of affairs will likely continue until, in the course of a few hundred generations, a structure will evolve which will be more in keeping with conditions then prevailing. Until then the author feels we must be content to continue to remove these structures when they become diseased.

This paper gives an excellent description of the lymphatic system in general and the subepithelial specialized portion in particular. It also presents an interesting theory as to the likely intended function of tonsillar tissue. For the present, however, when tonsils appear to have become diseased, their removal is the only solution.

Steele and Anderson[21] state that, although tonsillectomy is generally considered a relatively safe procedure, pulmonary and other complications occur more frequently than we physicians should like to admit. Literature is reviewed showing that atelectasis and pulmonary abscess not infrequently complicate tonsillectomy performed with general anesthesia. As aspiration of pharyngeal contents is favorable to pulmonary complications, they observed a series of 129 patients and found that 96.9 per cent were found to have measurable amounts of bloody secretion in the tracheobronchial tree. To overcome this, they outline a method of performing laryngoscopic tracheobronchial suction immediately after operation and present measures designed to minimize the possibility of aspiration during tonsillectomy and adenoidectomy with the patient under general anesthesia.

Comment: The authors seem unaware that certain of the papers they reviewed caused the tonsillectomists concerned to alter their technique greatly, with resultant change in the incidence of pulmonary complications. To prevent pulmonary complications following tonsillectomy and adenoidectomy under general anesthesia, two requisites are necessary: the cough reflex must not be diminished by preoperative sedation and the anesthesia must be so well timed that the patient clears his tracheobronchial tree by coughing before he is allowed to leave the operating table.

Coombs[22] reports the study of 653 consecutive cases of tonsillectomy and adenoidectomy to discover the incidence of postoperative bleeding and found that the critical period lies between the third and eighth days. The incidence of bleeding from the adenoids was far greater than from the tonsil fossa.

Taylor[23] describes the sudden death of a seven-year-old Negro shortly after tonsillectomy in a great hospital where adequate preoperative examination had been made and where every facility was available. The operation was performed under endotracheal oxygen and ether. Profuse bleeding from each tonsil fossa and from the nasopharynx was encountered, necessitating ligatures in each fossa and a nasopharyngeal pack which successfully controlled all bleeding. After tracheal aspiration, the endotracheal tube was removed and the patient moved out of the operating room to await recovery. Stertor noted at this time was attributed to a combination of a large, thick tongue and the postnasal pack; however, the patient's color was good, and a metal airway did not seem necessary.

During the postoperative period the child was being observed at regular intervals by either a nurse or an anesthetist, but she was not under constant observation by any one person.

A student nurse noted that respiration was very labored and cyanosis of the lips and nails was quite evident. She called the anesthetist, who found that respiration had ceased entirely when he arrived. He immediately started artificial respiration with face mask and positive pressure apparatus; however, respiratory exchange did not seem to be sufficient by this method so the endotracheal tube was replaced. After it was noted that respiration had ceased, probably five or 10 minutes after the patient left the operating room, almost another minute passed before the airway could be restored. There was no obstruction by blood or secretions. Good expansion of the chest was obtained with each compression of the breathing bag. There was no improvement in the cyanosis, and the heart ceased to beat at approximately the time the airway was restored. A general surgeon was summoned from an adjoining operating room. The chest was opened and manual massage commenced. In spite of every measure — all of which are described — cardiac failure occurred about four hours later. The autopsy findings are recorded in detail.

No claim is made that the death of the patient was the result of factors other than anoxia from obstruction of the airway.

The following are some of the measures suggested to prevent further similar episodes: *1.* There should be adequate postoperative observation, especially so when a postnasal pad has been inserted. *2.* The airway must be kept free and open by whatever means are necessary.

Bryant[24] reports a case of retropharyngeal abscess in a six-year-old child. A photograph of the Roentgenogram showing the condition is given. It was felt that the abscess was secondary to an infection in a regrowth of adenoid tissue.

IRRADIATION WITH THE NASOPHARYNGEAL APPLICATOR.

Bilchick and Kolar,[25] from the Department of Otolaryngology, Columbia Presbyterian Medical Center, report an analysis of 468 cases on whom the nasopharyngeal radium applicator was used for the prevention and treatment of nasopharyngeal and nasal complaints.

An applicator was used in each side of the nose for 12 minutes for three treatments with an interval of two to four weeks between treatments. The turbinates were shrunk before application and the applicators were crossed in front of the columella.

In a number of their cases otitis media flared up on the eleventh to thirteenth day, indicating the height of the local erythema. Dryness of the throat, irritation of the throat and flare-up of previous otitis media were seen in almost half of their patients.

A high proportion of a group of children who received this treatment were greatly benefited. They considered the treatment beneficial for conductive deafness in children, but noted that allied symptoms involving the ears, nose or throat were benefited in only a few cases.

The writer of the resumé has observed the effects of such treatment over an even larger series of cases and has only very rarely recorded the irritation above described, due, he believes, to the fact that the applicators are not crossed in front of the columella and hence do not press tightly against the Eustachian cushions.

Lederer,[26] impelled by reports of enthusiastic acceptance, as contrasted by dire warnings of the dangers of irradiation of the nasopharyngeal lymphoid tissue, dispassionately discusses the two sides of the problem.

His most interesting contribution is a lengthy quotation from T. J. Wachowski, Professor of Radiology at the University of Illinois. In common with many radiologists, he, on the whole, prefers properly controlled X-ray therapy. (*Comment:* If there is an argument against the use of the radium applicator in the nasopharynx the argument would seem to be at least equally strong against the use of X-ray therapy which is not properly controlled.)

Lederer's summary is as follows:

1. Considering all of the available evidence, irradiation of the nasopharynx by the standardized techniques is without danger. It should, however, be used with restraint

and in competent hands, because of our incomplete knowledge of radiobiology and the empiricism of the present dosage.

2. Its use is not a substitute for surgery and should be confined to those cases of conduction deafness associated with the proven presence of lymphoid hyperplasia in and about the Eustachian tube orifices.
3. The syndrome of transitory hearing loss, allergic manifestations and infection in the nose, sinuses and/or nasopharynx, must be recognized and treated accordingly, in addition to irradiation
4. The importance of history taking in relation to previous irradiation is stressed because of the possibility that a repeated series of treatments may produce untoward tissue changes.
5. The warning of the danger to the careless technician himself must be respected
6. The use of irradiation in irreversible hearing deficits is to be deplored and discouraged.
7. We are agreed that in competent hands no great advantage is to be had from R over X irradiation. As a matter of fact, the simplicity of obtaining and carrying out the first form of therapy may eventually lead to the harm we wish to avoid
8. The increasing prevalence of the routine postoperative irradiation of the nasopharynx is purely an admission on the part of the otolaryngologist that this operation is technically inadequate
9. Histologically, all stages of degeneration in the lymphoid tissue may be demonstrated following irradiation. This is then followed by varying degrees of reparative change from granulation tissue to localized areas of ossification in occasional cases No further metaplastic change has been noted, nor has there been any alteration of the surface epithelium

Rubin, Kelly and Kinkle,[27] in a carefully monitored series of treatments with the 50 mg. monel metal nasopharyngeal radium applicator, report that total body irradiation did not exceed 6 mr per treatment for the physician and assistant. The tolerance dose for the whole body is 300 mr per week. Average exposure of the entire length of both index fingers of the physician was 26 mr and of several fingertips 50 mr per treatment. Pending official recommendation, they have adopted a figure of 600 mr per week as permissible exposure for hands and fingers. In accordance with this value, the maximum number of treatments which one physician may administer weekly by their technique is 12, fingertip and not whole body exposure being the limiting factor.

Their results suggest that generally accepted recommendations as to the number of treatments physicians may administer without endangering themselves are too high for safety.

Lead shields or other accessories for the radium applicator or modifications of its construction might reduce the dose delivered to the physician's fingertips and thereby permit a larger number of treatments.

The technical part of this well-conducted study is followed by a discussion of many practical details of technique and also by the instructions which the authors hang on the wall over the radium container for all personnel handling radium to follow.

POLIOMYELITIS AND TONSILLECTOMY.

Cunning[28] reports the completion of a four-year nationwide survey conducted by the American Laryngological, Rhinological and Otological Society, Inc., in an effort to determine whether or not there is any relationship existing between poliomyelitis and tonsillectomy and, if so, whether it be casual or coincidental. He publishes eight tables summarizing the results of the survey of 1949.

During the four years, 36,678 cases of poliomyelitis and 93,379 cases of tonsillectomy were studied and in these series only seven cases of bulbar poliomyelitis followed tonsillectomy — an incidence of 0.000073 per cent.

The committee, therefore, concludes that it fails to see any causal relationship existing between poliomyelitis and tonsillectomy, nor, up to and including the present time, have they read any published reports from other sources that offered any conclusive proof that tonsillectomy does predispose to bulbar poliomyelitis As they have said in previous years, they see no reason why tonsillectomy should be indefinitely postponed simply because the Summer months are the months wherein poliomyelitis is prevalent, however, they do not advise any elective surgery being done during any epidemic, regardless of its nature

Anderson, Anderson Skaar and Sandler,[29] from the School of Public Health, University of Minnesota, aided by a grant from the National Foundation for Infantile Paralysis, present a short, clear, well-analyzed study of an outbreak of poliomyelitis in Minnesota, in 1946, which supports the contention that poliomyelitis occurring in the first month after tonsillectomy is more apt to be of the bulbar type than is infection occurring under other circumstances

The article deserves careful study in its entirety It comments fairly upon the conclusions of Kinney and Cunning and avoids the faults the authors find in the data and arguments from and by which those conclusions were reached

In 1946, Minnesota experienced an extensive outbreak of poliomyelitis involving 2,881 officially reported cases and 226 deaths Detailed epidemiologic histories were obtained from 2,709 cases The outbreak reached its peak late in July in Minneapolis and early in August in the remainder of the state. The occurrence of the poliomyelitis cases by week of onset is shown in the first table

In July, as it became apparent that a large number of cases of poliomyelitis could be expected, a warning was issued regarding the possible risk of tonsillectomy, and physicians and hospitals were urged to defer such operations if possible Many hospitals by formal action placed a ban on all but emergency cases; others discouraged operations The figures and comments provided definitely support the author's conclusion that the number of children who were tonsillectomized and thus could develop poliomyelitis within a month after operation was reduced to virtually zero between the middle of July and the end of September.

Of the 2,709 cases on which histories were obtained, 18 had had tonsillectomy or adenoidectomy during the previous month, 10 during the second preceding month and eight during the third preceding month. Tables show *a.* the age, sex, date of operation and of onset, and type and outcome, and *b.* the breakdown by type of infection of each of these 19 cases. It is apparent that not only were there more cases in the group developing infection within a month after operation but, also, that 12 of the 19 (63.2 per cent) were bulbar as contrasted with only four bulbar out of 18 cases (22.2 per cent) in the second and third months. During the entire outbreak, 20 per cent of all patients and 11 per cent of those under 12 had bulbar infection. These data are in keeping with those previously recorded in showing an undue concentration of bulbar type among those most recently tonsillectomized.

Other analytical tables enable the reader to follow the authors' arguments that rates based on all children developing poliomyelitis, regardless of age and irrespective of the period of the outbreak, are meaningless since the risks of developing the infection vary so greatly at different ages and stages of the epidemic cycle; and that the only truly valid comparisons are those based on groups of equal age and periods of exposure.

The authors make a statistical analysis which bears every evidence of statistical fairness and come to the final conclusion that the risk of developing poliomyelitis was at least three times as great among those undergoing tonsillectomy as among a comparable group not undergoing this operation and that the risk of bulbar infection was 11 times as great.

One of the editors of the *Journal of Pediatrics,* L. F. Hill,[30] in his column discusses tracheotomy in bulbar poliomyelitis. He reminds us that fatalities in poliomyelitis are almost exclusively due to the bulbar and respiratory paralytic forms of the disease; however, there is a difference of opinion in treating this condition and the controversy ranges around the value of tracheotomy. The editor mentions the work of the Minneapolis group in the 1946 poliomyelitis epidemic and how they undoubtedly saved many lives by means of tracheotomy. He also reports the work of Galloway and Seifert and shows how they favor tracheotomy. On the other hand, he says that Stimson, from a large New York hospital, stated that cases where tracheotomy was indicated had not been encountered. Expertness of the staff in caring for bulbar poliomyelitis cases did away with the necessity for tracheotomy.

The editor shows that in his own group of cases, in Iowa, in 1949, tracheotomy definitely saved several lives; however, he admits that it will take many more reports before the issue can be determined. His conclusion is that fatalities from bulbar poliomyelitis can be reduced by means of expertly trained and equipped personnel with adequate facilities in all areas where poliomyelitis is epidemic, whether or not tracheotomy is employed.

MOUTH AND THROAT INFECTIONS.

Smith and Bloomfield[11] have made some interesting studies on the development of the bacterial flora of the throat in newborn babies. They found that the pharynx is always sterile at birth; however, as soon as the infant nursed, at about 24 hours of age, a variety of bacteria appeared, the most constant being nonhemolytic streptococci. The common pathogens of the upper air passages, such as hemolytic streptococci, pneumococci, meningococci and so on, were definitely absent, because no clinical infections had taken place as yet.

There was no striking difference between breast fed and bottle fed babies in the small group observed.

Another feature of the investigation was the effect of penicillin on the bacterial flora. Five cases were given repository penicillin as follows: *A* received 150,000 units every 24 hours for four doses beginning at four hours after birth. *B* received two doses of 150,000 units every 24 hours apart, commencing four and one-half hours after birth. Cases *C, D* and *E* each received a single dose of 150,000 units penicillin at four and one-half, three and 12 hours after birth, respectively. In each case it was but a few days after the cessation of the administration of the penicillin before bacteria were cultured from the pharynx. Then nonhemolytic streptococci were found just as in those cases which were not given penicillin.

Haffner, Neter and Rubin[32] made studies of the upper respiratory tract in children to see the effect of penicillin in producing a predominant Gram-negative bacillary flora. Observations were carried out on 90 chidren, all of whom received intramuscular penicillin. In addition, some received sulfonamides as well, and a few were given streptomycin. All but 16 cases showed a conversion to Gram-negative bacillary flora in the throat and nasopharynx. The administration of sulfonamides in conjunction with the penicillin did not materially alter the change in flora.

From a clinical point of view it was noted that those children whose upper respiratory flora became predominantly Gram-negative bacilli had a more severe illness, and also it was of longer duration.

Two cases of acute suppurative infections of the salivary glands in the newborn are reported by Shulman.[33] The first case was an infection in the left parotid gland in a seven-day-old baby. The other patient was three weeks old and had an infection in the right submaxillary gland. These infants had a fairly large tense swelling over the respective glands, and there was an elevation in temperature and the white blood count. Gentle pressure on the swellings produced pus in the mouth in each case. Cultures of this grew hemolytic staphylococcus aureus.

The treatment consisted of adequate doses of intramuscular penicillin and the swellings disappeared completely in a few days.

Rook and Brand[34] report a case of unusual infection in the tonsils caused by Candida Krusei. This fungus infection is rare, the common one being Candida Albicans or "thrush."

Their patient was an 11-year-old boy who showed irregular, elevated white patches on his tonsils. These patches were stony hard and it was necessary to break off pieces with forceps for culture study. Cultures revealed streptococci. Because tonsillectomy was refused, the boy was hospitalized, and after nine months the entire medicinal therapeutic armamentarium was exhausted with no change in the condition of the tonsils. Operation was then resorted to and he was discharged from hospital in two days. One year later there was no recurrence of the fungus infection.

Geiger and Marshall[13] state that diphtheria for long was considered to be the ideal textbook disease. It was an infection which produced a toxin. There was an antitoxin to combat the disease, and toxoid to prevent it. The Schick test told of those who were susceptible to diphtheria, and the virulence test was a laboratory method of telling the toxigenicity; however, the disease too often has failed to follow the textbook.

Diphtheria is known to be an infection and also an intoxication. A certain strain of diphtheria bacillus may be infectious but a poor producer of toxin, and another strain may show the opposite characteristics. Because of this, the Schick test may be negative, for it is only a test for threshold level of antitoxin, and yet the patient contracts diphtheria when the bacilli are exceptionally active in the production of toxin.

Toxoid may produce various antitoxin responses in the individual, and so again he may contract the disease, depending on the toxigenicity of the organism; however, the evidence still warrants the use of toxoid in children, as it reduces the number of cases of diphtheria and certainly the number of serious or fatal cases.

The virulence test takes three days to a week to perform and is probably useful only for the records. It is uncertain with those infectious organisms which lack any marked ability to produce toxins.

The Schick test, toxoid and antitoxin apply only to the intoxication, but it must be remembered that diphtheria is an infection as well.

The toxin in diphtheria affects peripheral and cranial nerves, rather than the central nervous system; however, the latter does occur, and Dolgopol and Katz[36] report five cases of encephalitis in diphtheria. These cases all showed C. Diphtheriae on direct smear or culture from the throat. Adequate doses of antitoxin and penicillin were given, but all died. Necropsy showed the typical lesions of encephalitis. The authors suggest that diphtheria infection affects the central nervous system more frequently than generally suspected; the central nervous system, also, was probably damaged by direct action of circulating diphtheria toxin.

SURGERY.

Bakwin[37] discusses psychic trauma of operations in children and reviews the recent literature. The removal of tonsils and adenoids is the commonest surgical procedure in children, and so this operation is blamed for most of the psychological damage which occurs from surgery.

Most children forget their hospital stay quickly, and are none the worse for their experience. Others, however, show alterations in behavior, characterized by fears, negativism, and family antagonisms. Many of these children had been poorly prepared for the operation, anesthetic and separation from the parents. Postoperative symptoms occurred at all ages, but the most frequent group was the one- and two-year-olds. These children are more dependent on their mother and home, and less able to understand what is taking place.

It is suggested that a kinder and more humane approach to anesthesia and surgery may help in preventing postoperative disturbances in children.

Heck, Hallberg and Williams,[38] from a study of 64 patients with antrochoanal polyp from the Mayo Clinic, believe that the antrochoanal polyp has important differences from the ordinary nasal polyp. While the ordinary nasal polyp is of allergic origin, the stimulus for growth of the antrochoanal polyp is of a different nature.

Contrary to the generally accepted opinion, the antrochoanal polyps do not occur predominantly in children, but affect the various age groups rather uniformly.

Nasal polyposis in a child can be a great distress to the attending rhinologist. Any measure which may possibly improve the therapy of this condition deserves attention.

Ruskin,[39] in a considerable number of adult patients, has found that nasal polyposis is amenable to conservative treatment by the use of solutions of procaine penicillin, 3,000 to 5,000 units per cc., when instilled into maxillary and sphenoidal sinuses.

Hayden, Pastore and Kundert[40] report the case of nasal septal abscess complicated by orbital cellulitis in an infant three and one-half years of age. When the patient was 17 days old the family physician prescribed nose drops for a head cold and nasal discharge. Two days later swelling of the left eye was noted. In spite of six daily injections of penicillin the condition became worse and when hospitalized the left eye was proptosed downward and outward with total loss of motility, and the septum was so swollen that the nose was completely blocked.

Intramuscular injections of 50,000 units of penicillin every three hours were initiated on admission and the septal abscess was incised and drained. The latter procedure was repeated on five other days. Although penicillin was administered for a total of 10 days, it apparently did not influence the course of the illness. The condition responded readily when sulphadiazine and dihydrostreptomycin were administered. Complete recovery ensued.

NEW GROWTHS.

Figi and Davis[41] maintain that nasopharyngeal fibromas may be expected to show signs of spontaneous regression before the age of 20. Seventy-three per cent of 63 cases previously reported by Figi completely disappeared before this age. The present paper reviews 51 additional cases. Few series report more than 10 cases. They are highly vascular, and locally invasive.

Other names are: nasopharyngeal fibroma, juvenile basal fibroma of the nasopharynx, fibroid tumors of the base of the skull, fibroangiomas of the nasopharynx and juvenile nasopharyngeal fibroma.

It is important to avoid confusion with innocuous fibrous tumors presenting somewhat the same appearance occasionally seen in the nasopharynx, nasal fossae and accessory sinuses of patients who have not reached the age of puberty.

The authors have never observed complete spontaneous disappearance.

Because of the locally destructive character of such tumors and their proximity to a number of vital structures the authors have not felt justified in marking time and idly observing them to see whether complete regression would occur.

They have been thought to predominate in males, but this series had no males. They never metastasize. Of the 51 cases, the age of onset ranged from nine to 17 years.

As a rule the authors *avoid biopsy* and they advise a combination of surgical diathermy and radiation.

Such procedures as splitting the soft palate and temporary resection of the maxillae in order to gain access to the tumor have gone into the discard.

A culcanite nasal speculum is used when the diathermy is applied through the nostrils.

They make the following conclusions:

1. The most satisfactory treatment for nasopharyngeal fibromas is a combination of electrocoagulation and interstitial radiation.

2. These measures may be used through the nares and transorally through the nasopharynx, but they are more safely and effectively applied through a transantral approach, with or without preliminary ligation of the external carotid artery.

3. With proper treatment a high percentage of nasopharyngeal fibromas can be completely eradicated or reduced to a small inactive remnant; in fact, by means of electrocoagulation and interstitial radiation applied transorally complete removal often can be accomplished in a single operation.

This excellent paper should be read in its entirety.

Sooy[43] discusses primary tumors of the nasal septum and provides 11 case reports. Such growths are rare. He names *the types that have been described. In many cases inadequate* removal of the original lesion resulted in recurrence. This necessitates subsequent radical resection which might not otherwise have been necessary.

As a result of studying the references, he adopted the following *procedures: 1. Small posterior polypoid lesions are* resected locally and the base cauterized. *2.* Small discrete anterior lesions are not biopsied. The surrounding mucosa and attached cartilage are removed under local anesthesia, leaving the mucosa on the opposite side attached. Vaseline gauze packing is placed over the defect for 24 to 48 hours and no cauterization is used. Complete epithelization occurs over the intact mucosa of the opposite side in four to six weeks. Vaseline is used to prevent crusting and there is no disability during this period. More extensive lesions are first biopsied and this is checked against the cytological smear findings. *3.* Large, benign, mucosa-covered lesions are resected intranasally with an adequate margin, and the edges of the septum are cauterized. General anesthesia is preferred because of the discomfort incident to adequate coagulation. When the lesion involves the bony septum the cartilaginous portion is excised *en bloc* and that attached to the bony portion is removed with punch forceps. There is sequestration of devitalized bone fragments from the margin during the healing process. Epithelization is complete in six to eight weeks. *4.* Large, fungating, ulcerated, malignant lesions are treated in a similar manner except that the maximum margin is obtained and the subsequent fulguration is very extensive. The period of sequestration and epithelization is consequently prolonged. 5. Posterior malignant lesions are resected intranasally with the entire posterior septum and most of the anterior part. The margins are thoroughly fulgurated. The extensive septal resection is carried out in order to facilitate subsequent observation for evidence of recurrence.

One of the patients was a white female infant, age three months. The infant had an intranasal mass since birth, which blocked both sides. During the past two months there had been considerable increase in the size of the mass, which had widened the nasal bones exteriorly. The nasal obstruction interfered with feeding, and there has been no recent weight gain. A biopsy taken one month previously was reported as showing a benign fibroma. The large intranasal mass arose from within the superior portion of the nasal septum. Its surface was smooth and spherical in shape and measured 2 x 2.5 cm. The base of the nose, exteriorly was widened, and there was a 0.5 cm. separation of the nasal bones.

Under general anesthesia a large, encapsulated, firm, fibrous mass was removed intranasally and subsequent healing was uneventful. The final pathological diagnosis was atrophy with glial tissue.

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THE STRIA VASCULARIS: REVIEW AND OBSERVATIONS.

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1. *Bibliographical Introduction.*

The earliest description of the stria vascularis is attributed by some to Huschke, and the structure is sometimes referred to as the stria vascularis of Huschke. Other workers credit Corti with being the first to describe it. Corti studied the stria in mammalians and as early as 1851 suggested that it might have some connection with the secretion of endolymph. Verifying Corti's description of the stria were Deiters (1860) and Hensen (1863). On the basis of studies of the cat's embryo, Böttcher (1870) was the first to furnish embryological data on the stria. Theories regarding the formation of the stria were *advanced by von Winiwarter (1870), Gottstein (1871)* and Waldeyer (1872) Retzius, in 1881, suggested that the stria represents a vascularized epithelium. This concept, about 50 years ago, was a subject for much debate and many investigations by well known names in otolaryngology. Shambaugh, in 1907, through embryological studies, placed the development of the stria on a firm foundation. This will be discussed in more detail later. Several comprehensive papers on the stria were written about 1900. They contained bibliographies on the subject up to the time of their publication. These included the basic works of Leimgruber and Shambaugh.

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2. *Embryology and Histology.*

As previously mentioned, about 50 years ago there wa debate as to whether or not the stria was a vascular lium. Winiwarter, in 1870, denied the epithelial struc the stria on the basis of the principle that the main teristic of epithelium is the absence of blood vessels. gruber discussed the presence of vascularized epithe the nonotic organs in mammals and tends to refute W ter's arguments.

Proponents of the epithelial origin of the stria were Kölliker, Corti, Deiters, Hensen and others. This the refuted by Gottstein, Baginsky and Katz. Leimgru proached the problem through studies of the embry development in the guinea pig and man. He conclud there are two entirely different tissues in the stria layer of epithelium, and the other a layer of connective these two layers not being separated by a basemen brane. The layers are so intertwined that they seem uniformly built structure. He concluded that the epi comes in contact with the vessels but takes no part i formation.

All of the preceding studies were analyzed and adde Shambaugh, who advanced what is now the generally a theory of the development of the stria vascularis. His were based on embryological specimens, and he de three distinct stages of development.

Stage 1: A simple row of epithelial cells develop the external wall of the cochlear duct, with a dis basement membrane dividing the epithelium from underlying connective tissue.

Stage 2: The development of a mesh-like layer the surface epithelium, with the disappearance o basement membrane and the beginning formation of vessels.

Stage 3: The adult form (see Fig. 5)

In studying the transition from Stage 1 to Stage 2, Shambaugh saw "protoplasmic processes" from the surface epithelium penetrating the entire stria and surrounding the newly formed blood vessels. By following these changes he concluded that the cells of the reticulum originate partly from the surface epithelium and partly from the underlying connective tissue. Some of the blood vessels of the stria developing under the surface epithelium were seen to be sheathed by the protoplasmic processes of the epithelial cells. Finally, he states that the stria vessels in the adult are derived from surface epithelium and that the stria vascularis is a true "vascular epithelium." He also admits that the cells of the deeper layers show more connective tissue character.

Fieandt and Saxén, in 1936, made a detailed report of the cytology of the stria based on histological studies in dog and man. They feel that the majority of the cellular elements of the stria belong to the chromophile group of cells. In the early stages of development, the cells of the stria contain glycogen. Kolmer described the resorption of glycogen from the cells as they matured. As the cells develop, they assume the characteristics of chromophile (pigment) cells, these cells being the vacuolized superficial cells of the stria. With advancing age, the chromophile cells become more and more prominent. Fieandt and Saxén felt, regarding the human stria, that there was no doubt as to the epithelial origin of the entire structure. They found, through studies of the histochemistry of the cells, that the pigment of the stria vascularis is formed by the transformation of the plasma and the nucleus. There is a definite relationship between the presence of the pigment and Golgi apparatus within the cell. The endolymph is formed in the basal portion of the cell and always inside of the Golgi apparatus. The secretion rises with the Golgi apparatus inside the cell, goes around the nucleus and is localized under the surface membrane. The drops penetrate individually the surface membrane of the cell. Cajal noted that the Golgi apparatus loses its staining reaction after the evacuation of the secretion. The interpretation and significance of the described findings will be discussed under "Function."

3. Function of the Stria Vascularis.

More recent studies of the stria vascularis have been centered about the functional and pathological changes. The various theories of the function of the stria may be divided into three groups:

1. Secretion.
2. Excretion.
3. Mechanical.

The majority of workers have conceded that the stria vascularis has a secretory function and plays a part in the formation of endolymph.

1. Shambaugh, in 1908, has described a modification of the surface epithelium of the stria into gland-like structures. Held confirms the presence of translucent vacuolized protoplasm with gland-like characteristics. These modified cellular structures have the name of "Glands of Shambaugh." The findings of Fieandt and Saxén offer further proof of the existence of these structures.

Additional support of the theory that the stria vascularis secretes endolymph came from Fraser, who studied deafmutes. He found that in cases where the stria was abnormal there was collapse of the cochlear duct. Kishi, also, in a study of Japanese dancing mice, found the only consistent abnormality to be degeneration of the stria. Saxen, in studying deafmutes, concluded that although the stria plays some part in the manufacture of endolymph, there is a similar process of secretion in the vestibular membranous labyrinth. Our findings confirm those of Saxen.

2. Wilkinson and Gray suggest that the function of the stria may be excretion — to remove waste products of cell metabolism from the endolymph and maintain the chemical constancy of the labyrinthine fluid.

Stacy Guild, in 1927, carried out experiments on the circulation of the endolymph. He followed the flow of the endolymph by injecting potassium ferrocyanide and iron citrate into the cochlear duct and sacrificing the animals at intervals of 45 minutes to 44 hours. Prussian blue granules were precipitated by using an acid fixative. Since there were few granules in the stria and many in the saccus endolymphaticus, he concluded that the endolymph is formed by the stria vascularis. The fluid flows toward the basal end of the cochlear duct and through the canalis reuniens into the sacculus, through this to the ductus endolymphaticus into the bulbous endolymphaticus, and leaves the membranous labyrinth by passing through the walls of the pars media of the bulb into the small blood vessels of this region. He compares the excretion of the endolymph through the walls of the bulb to the flow of the cerebrospinal fluid through the arachnoid villi.

3. Kolmer suggests that the variations in the filling of the strial vessels may influence the form and volume of the cochlear duct. Gray suggests that sound may, by a nervous vasomotor reflex, cause an increase in the flow of blood to the inner ear.

Belemer suggests that the stria is an accommodation mechanism which responds directly to stimulation by sound. He feels that there may be a direct mechanical response which works by the changes in the distention and constriction of the strial vessels to adjust intracochlear fluid pressures of the basilar membrane so that it can best perceive sound. Szasz called the strial system of vessels the "buffer system" of the labyrinth. This idea was elaborated by Agassiz, who feels that the circulation of the stria is constant and that arteriovenous arches of the vessels of the spiral ligament act to keep the blood flow to the rete of the stria constant as well as remove the rhythm of the pulsations of the blood. He states that it is this arteriovenous arch which protects the endolymphatic system from variations in pressure as a consequence of circulatory changes rather than any mechanism in the stria itself.

Fieandt and Saxén suggest the possibility of an automatic regulating mechanism normalizing the production of endolymph to preserve a constant endolymphatic pressure. If the latter increases in the inner ear, this is noticed at once with the help of the supporting fibres of the chromophile cells and by the blood vessels. Mechanical compression of the blood vessels follows with a diminishing production of endolymph, resulting in the restitution of the pressure in the cochlear duct. With decreased pressure, the contrary mechanism is followed, a dilatation of the vessels increasing secretion and restoring the endolabyrinthine pressure.

Still other workers feel that the constancy of the endolymphatic pressure and chemical composition is dependent, at least in part, on the proximity of the endolymphatic fluid with the perivascular lymph spaces of blood vessels and nerves which form an important relationship between the intracranial space and the membranous cochlea.

In early reports Prenant described cells in the stria which he felt resembled muscle cells. These findings were never confirmed by other workers.

4. Pathology.

As in other structures of the temporal bone, the study and interpretation of the microscopical sections have to be correlated with the clinical manifestations of various diseases. Thus there arises much debate as to whether or not many of the findings so clearly described are not artifacts or postmortem changes. The interpretation of thrombosis of strial vessels, hemorrhage into the stria, dilatation of the blood vessels, and so forth, have to be made with some reservations; however, for want of a more accurate method of study, such descriptions must be considered, judged, and some attempt made to draw conclusions as to their significance. Mosher, Alexander and Saxen have all warned against too much skepticism, as the other extreme, in the interpretation of microscopical findings.

From the earliest descriptions of the stria and up to the present time, many authors have reported pathological changes in the stria. A summary of the strial changes in deafmutism up to 1926 was made by Sakūtarókano. The main finding was a proliferation of the epithelium Other changes noted were duplication of the stria and separation of the stria from the spiral ligament. In later studies, Carruthers describes the stria as having relatively few blood vessels and being less cellular than normal. Fraser, in studies of deaf-mutes, describes the collapse of Reissner's membrane in cases where the stria is degenerated or absent Saxen, in similar studies, found the stria normal in one ear and normal in some sections and atrophic in other sections of the opposite ear Other pathological changes in the two ears were similar

Hallpike and Cairns, in two specimens, describe the human temporal bone changes in Menière's disease One specimen shows a normal stria and the other shows degenerative changes

5 Specimens and Material Studied

In the selection of the material studied there was a large series of temporal bones from various animals Human temporal bones were not studied because of the feeling that many of the changes involved were postmortem rather than pathological We studied specimens in which we could show the normal stria, the stria in degenerative inherited conditions, and the stria of animals with acoustic trauma It was decided to study the guinea pig chiefly The normal specimens were well stained with hemotoxylin eosin stains and showed all structures to be normal For the second group, congenital changes, waltzing guinea pigs were used These animals tend to run in circles and become deaf The changes in the other structures of the temporal bones of these animals were described by Lurie The deafness and circling is a Mendelian recessive characteristic. Several specimens of Dalmatian dogs also were included

To show the changes of acute acoustic trauma, the temporal bones of guinea pigs that had been exposed to loud sounds at various intensities and frequencies were used The changes in the organ of Corti have been reported in a previous paper.

The specimens studied included the temporal bones of: two normal guinea pigs, four waltzing guinea pigs, three guinea pigs that had been exposed to acoustic trauma, two Dalmatian dogs.

The studies were carried out in the following manner: Every fifth slide of the continuous series of temporal bone sections was studied. In case of special interest every slide was examined. The results were tabulated on charts in the manner illustrated in Table 1.

The most consistent abnormalities found in the stria were absence of the basement membrane, and atrophy of the cells with complete loss of the normal architecture. The organ of Corti showed changes varying from beginning degeneration of the outer hair cells to complete destruction of the entire structure. Reissner's membrane in the Dalmatian dogs was collapsed and adhered to the stria.

6. *Observations.*

An attempt was made to correlate the changes in the stria vascularis with the appearance of the organ of Corti and the contour of the scala media. In the specimens of the waltzing guinea pigs, there was marked atrophy of the stria with a scala media of normal contour (see Figs. 1 and 2). This failure of the scala media to collapse indicated that only a very small portion of the stria is necessary to secrete a sufficient amount of endolymph to preserve the normal contour of the scala media, or that there are other sources of secretion of the endolymph. In cases of complete collapse of the scala media with atrophy of the stria, as in the Dalmatian dog, the utricle and semicircular canals had a normal amount of endolymph in them, raising the question again of whether the bulb of the ductus endolymphaticus or the region around the sensory neuroepithelial plaques of the cristae and maculae may not have an important secretory function.

In the waltzing guinea pig, several specimens showed complete degeneration of the stria with a normal organ of Corti

TABLE 1 — SIGNIFICANT FINDINGS.

Turn	Organ of Corti	Width	Extent	Vessels	Color	Pigment	BM*	Reissner's Membrane	Remarks
Basal									
2									
3									
4									
5									

*BM—Basement membrane.

Pigment—Presence of vacuoles Presence of pigment granules

Cells—Polarity, architecture.

Color—Ability of stria to take stain, excluding pigment granules.

Vessels—Number, size, endothelial cells, thrombosis

Extent—Spiral ligament (outer wall of scala media) occupied by the stria.

Width—Thickness of stria from BM to the surface.

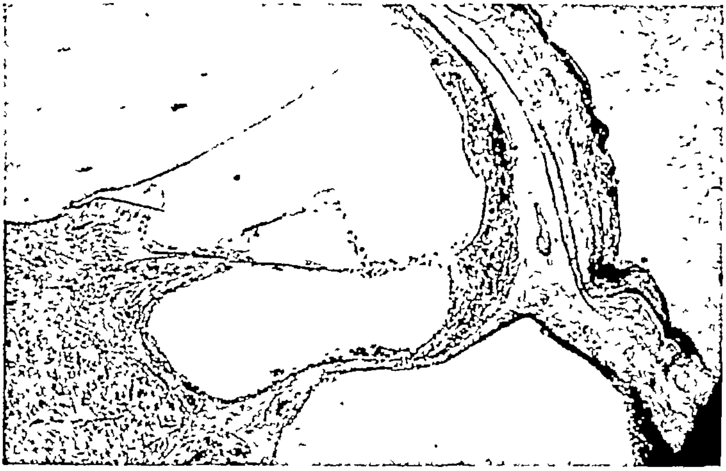

Fig. 1 Guinea pig; trembler; fourth turn. Note thin atrophic stria vascularis, degeneration of organ of Corti, normal contour of scala media.

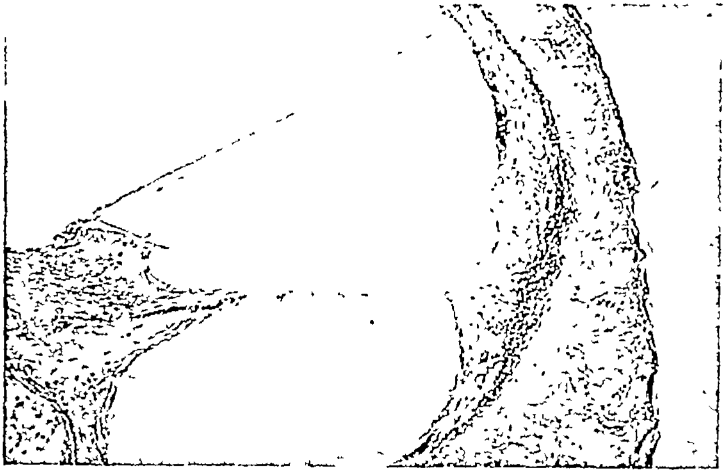

Fig. 2. Guinea pig (five years old); waltzer; third turn. Note: (1) Organ of Corti; (2) absent, stria vascularis atrophic; (3) normal contour of scala media.

only at the helicotrema (see Figs. 3 and 4); but in all examined specimens, where there was an abnormal organ of Corti, there were degenerative changes in the stria (see Figs. 1 and 3). In the temporal bones that had been subjected to acoustic

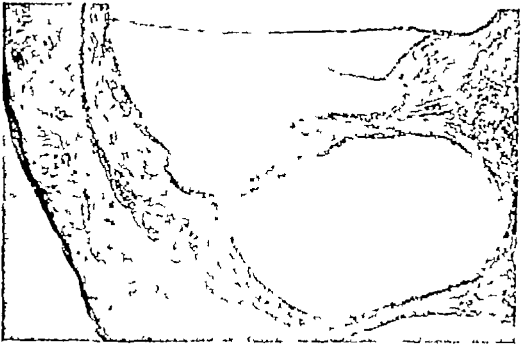

Fig. 3 Guinea pig waltz [illegible] turn (same specifications as Fig 4) Note (1) Stria vascularis [illegible] within normal limits (2) organ of Corti undergoing degener [illegible] and normal contour of scala media.

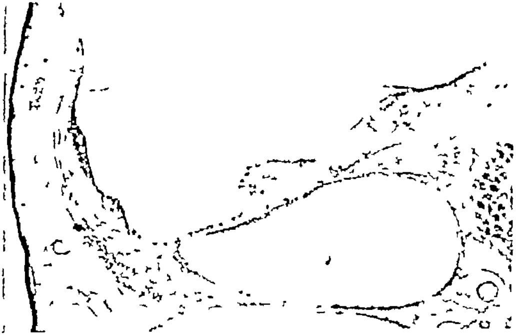

Fig 4 Guinea pig waltzer baby fourth turn specimen same as Fig 3 Note (1) Stria vascularis thin and atrophic (2) organ of Corti normal, (3) normal contour of scala media

trauma there was destruction of the organ of Corti with the stria normal. In animals with acquired and inherited changes of the organ of Corti and stria the scala media maintained its normal contour.

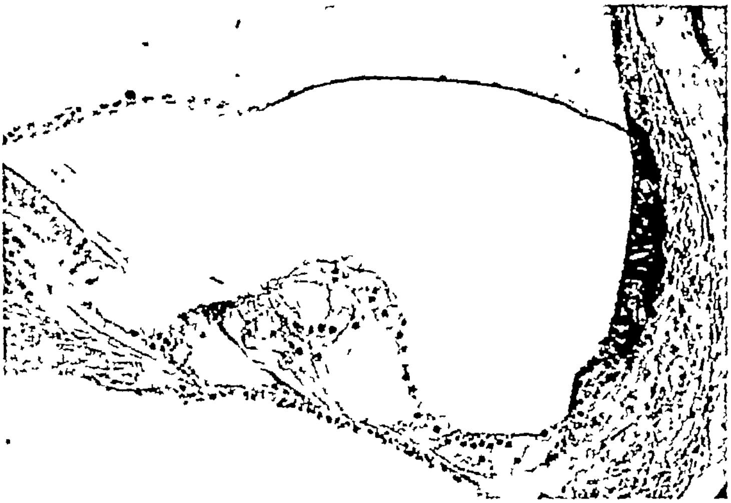

Fig. 5 Guinea pig, normal.

7. Summary.

A review of the literature on the stria vascularis was made. The subject is presented under the headings: bibliographic introduction, embryology and histology, function, pathology, material studied, and observations. From reports in literature, no constant abnormality of the stria could be found to account for any of the clinical entities known. Original studies of animal specimens, some with inherited and others with acquired abnormalities, are reported.

8. Conclusions.

In specimens with inherited changes, abnormalities in the organ of Corti were accompanied by changes in the stria, whereas changes in the stria were not necessarily accompanied by abnormalities in the organ of Corti.

In acoustic trauma the organ of Corti may be destroyed with a stria of normal appearance.

In the evaluation of agonal and postmortem changes we believe that one can accept changes as pathological in the stria and in the organ of Corti if one of these structures appears well preserved.

In the animal specimens studied no constant correlation between the changes of the stria vascularis, the contour of the scala media, and the organ of Corti could be found.

We express our thanks to Dr. George Kelemen for his advice and translations of the German articles, and to Jeanette Loessel for her aid in the compilation of the bibliography.

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PROGNOSIS IN TRAUMATIC PERIPHERAL FACIAL PARALYSIS.*

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The prognosis in lower motor neuron paralysis, of traumatic origin, of the VIIth nerve is difficult at best. For that reason we submit the various factors involved. These are: first, the history of the paralysis; second, the physical examination; and third, the electromyographic study.

1. *History* of the injury, if operative, is of no value, as the operator always insists that he has not been near the nerve and has no knowledge of when or how it was injured.

In 28 patients there were but two who had operators with knowledge of where, how and to what degree the nerve was injured. One of these had coagulated the severed ends for bleeding, which gave a bad prognosis for spontaneous recovery and called for nerve grafting. Gunshot wounds, basal skull fractures and other trauma such as automobile accident lacerations give no prognostic data, except in patients with a large tissue loss and obvious anatomic interruption of the nerve.

2. *Physical examination* is of value when the following factors are considered:

  a. Presence or absence of emotional control (which is lost first).

  b. Presence or absence of voluntary motion (which is next to disappear).

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c. Presence or absence of muscle tone which is the last to disappear. This is most important and has proven to be the most reliable factor available. If it be lost, then interruption of the nerve's continuity is obvious and intervention becomes necessary. We have held to the plan of waiting surgery for six months on a patient who maintains good tonus and our results justify this.

d. Response to faradic stimulation has not helped us.

The end-result in spontaneous recovery is more quickly attained and shows as good ultimate results as surgical repair. Both the surgical and nonsurgical recoveries show contractures, tics and associated movements having no relation to the *time of recovery nor the time or type of destructive or restorative* surgery. This is disputed by advocates of early surgical intervention. If tonus is poor, one must guard against muscle stretching by suitable support of the musculature. Should too long a time elapse before functional return it may be necessary to restore facial symmetry by fascial supports. Galvanic stimulation of the facial muscles is of questionable value.

Reliance on the evaluation of the above three factors noted *in the physical examination resulted in error in one patient; i.e.,* we thought function would return without surgical repair. It did not and surgery was needed.

3. The real purpose of this presentation is to call attention to the value of electromyography, reported in some detail by Wedell, Feinstein and Pattle[1] in *Brain,* 1944. It is a distinct aid but should *never* be relied on to the exclusion of the physical examination.

Electromyography is based on the fact that when muscles contract visibly or invisibly on voluntary or reflex effort they generate a type of current *called a motor unit action potential.* It is 100 microvolts to 1 millivolt in intensity; lasts 5 to 10 milliseconds and varies from 5 to 30 waves per second. A motor unit consists of the anterior horn cell with its axon and the 100 to 150 muscle fibres the axon innervates.

A denervated muscle shows individual muscle fibre twitching or fibrillation in a rhythmic manner. This does not occur in the facial musculature for from *12 to 14 days after* denervation. Voltages resulting from this fibrillation are known as *fibrillation action potentials.* They show mono- or diphasic spikes up to 100 microvolts in amplitude are 1 to 2 seconds in duration and repeat a rate from 2 to 10 per second.

These voltage responses are made visible on a cathode ray oscillograph and audible on a loud speaker. The visible responses may be photographed for permanent records.

Dr. Eugene Webb, of the University of California Neurosurgical Department, who has done all the electromyograms for me, has given me great help by furnishing the following outline by which one interprets the electromyographic studies:

"In summarizing the usefulness and technique of employment of the electromyograph, one may say that the key question concerning injuries of a peripheral nerve such as the facial generally may be answered more accurately and rather earlier than by clinical means alone. The question in one's mind, of course, is, has there been complete anatomical interruption, or has there merely been a so-called physiological block of nerve impulses to account for the apparent and complete paralysis of the involved muscles? In actual practice one tends to find with facial nerve lesions admixtures of varying degrees of these two possibilities. Generally there is some anatomical interruption and some physiological blocking to account for the clinically complete paralysis.

"Electromyographic studies may reveal occasional motor units under voluntary control which are incapable of producing a detectible movement clinically, but which are incontrovertible evidence of continuity of at least some of the nerve fibres. Occasionally no motor action potentials under voluntary control are encountered, but they will appear with mechanical stimulation of the needle movement and from a prognostic standpoint have the same significance.

"If no motor action potentials are noted, either with voluntary effort or with mechanical stimulation, the presence of fibrillation action potentials *in large numbers* in all muscles tested normally suggests complete or near complete mechanical interruption.

"Not infrequently, cases are encountered in which there are no motor units appearing under any circumstances, but in which fibrillation action potentials, though present, are *few* and *quite scattered.* Then it is our feeling that this pattern indicates that there is no complete anatomical interruption and a further period of observation is in order."

We had arrived at a similar conclusion without the electromyograms but welcome them as an aid in determining the validity of our withholding surgery.

There have been four patients we have observed in whom the clinical findings regarding emotional control, voluntary motion and the presence of muscle tone indicated the possibility of spontaneous reinnervation without surgery. We were supported by the electromyographic studies of Dr. Eugene Webb and report recovery in all.

CASE REPORTS.

Case 1: Postoperative facial paralysis (right radical mastoidectomy done elsewhere on Sept. 19, 1949)

When first seen, Sept. 24, 1949, there were no voluntary nor emotional movements, but there was some muscle tone The surgeon was at a loss for an explanation of how or where the nerve lesion had occurred. We advised against intervention Two months later he had electromyograph studies by Dr. Webb, who reported "electromyography was carried out on the frontalis and orbicularis oris No motor action potentials, voluntary or involuntary, were encountered in any needle position. Fibrillation action potentials indicative of anatomical interruption were encountered, but *in extremely small numbers*"

By Dec. 21, 1949, "direct muscle reflexes were present in all muscle groups supplied by this nerve and were responsive to percussion."

By April, 1950, he had progressed to complete recovery of voluntary motion but had tics, contractures and associated movements characteristic of all lesions in continuity. Taste on the anterior two-thirds of the tongue on the damaged side has returned, tearing has ceased. The frontalis has not returned and never will.

Case 2: The second patient had a labyrinthotomy and radical mastoidectomy on Aug. 9, 1950. He had good muscle tone; impaired but not lost taste, plus marked tearing on the operated side. There was questionable action in the orbicularis oculi.

On Oct. 12, 1950, electromyography was done by Dr. E. Webb, who reported "electromyographic study revealed *minimal fibrillation action potentials* in the frontalis and orbicularis oris. In the frontalis one small motor unit voluntary potential was observed. In other muscles tested, occasional involuntary action potentials were observed." This did not show a complete lesion of interruption of continuity and his recovery by mid-January, 1951, bore out the findings.

Case 3: This patient was similar and recovered similarly.

Case 4: The fourth patient reported had had a radical mastoidectomy by me in 1933 when he was eight years of age. A massive cholesteatoma was removed. Until the last two years the cavity gave much trouble from exfoliated epithelium and the tubal region was and is wet, though it is no longer troublesome. Nov. 26, 1950, he noted trouble in controlling his lips while brushing his teeth. In 24 hours he noticed difficulty in moving the face on the operated side. In 48 hours it progressed to present status of Nov. 30 when examination showed:

1. Good muscle tone.

2. Questionable flicker of voluntary motion at corner of mouth.

3. Tearing moderately increased.

4. Direct muscle reflexes not active.

5. Taste sense absent on anterior two-thirds of tongue on impaired side.

The mastoid cavity was clean and no erosions could be found along the course of the nerve.

The slow development of the paralysis suggested an ischemic lesion, so it was decided to wait until electromyograms could be done. This was done on Dec. 6, 1950, which was 11 days after onset: "The report showed motor action potentials in all muscle groups supplied by the VIIth on the impaired side. There were no fibrillation action potentials." This finding is typical of an ischemic paralysis without interruption of continuity, so intervention was not advised.

Recovery proceeded rapidly until Dec. 21, 1950, when he was last seen—by which time voluntary motion was nearly completely recovered. There were a few fibrillary twitchings at the corner of the mouth; associated movements were minimal and he had regained the use of the frontalis.

It must here be stated that Sullivan[4] and Violé[5] (personal communication after reviewing this brief paper) do not hold with the opinion that electromyography is of value. Sullivan states, "The interpretation of electromyography results is stricely limited in Bell's palsy." He makes no comments on traumatic peripheral VIIth nerve lesions.

CONCLUSION.

It is felt that electromyography in traumatic lower motor neuron disease of the VIIth cranial nerve is an aid in the prognosis of recovery without surgical intervention. The reports of electromyography are only as good as the doctor making them.

Three cases of surgical paralysis and one of ischemic paralysis complicated by an old radical mastoidectomy are presented in furtherance of this view.

The purpose of this presentation is to advocate the full use of clinical observation plus electromyography in reaching a conclusion as to when to operate or defer operation in patients suffering from peripheral traumatic facial paralysis.

It is our considered opinion that more nerves will be saved to resume their normal function when the above factors are carefully evaluated than by hasty surgical intervention by the average operator, whatever method he may use to restore physiological function, be it decompression, rerouting with end to end anastomosis or nerve grafting.

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EXPERIMENTAL POLIOMYELITIS.

Use of Human Gamma Globulin in Tonsillectomized Monkeys.

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The viruses of poliomyelitis produce widespread subclinical infection in man, particularly during epidemics. They may be detected in recognized cases of poliomyelitis, in mild suspected illnesses, and in healthy individuals.[1] Neutralization tests on human sera show an extensive immunity among the general population. "Present figures indicate that there are probably at least 100 to 1,000 poliomyelitis infections for every paralytic case."[2] It has been recognized for many years that the severe cases of poliomyelitis are often associated with stress such as pregnancy, strenuous exercise, as well as with seasonal and climatic factors. More recently, the inoculation of antigens for prophylactic immunization and surgical procedures, particularly those involving the mouth and pharynx, have come into prominence as factors closely correlated with the paralytic forms of the disease.

Inasmuch as human gamma globulin has been shown to contain neutralizing antibodies against the three known types of

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poliomyelitis virus,[3] it is of great interest to learn whether concentrated globulin processed from large pools of human donors would modify or prevent the serious forms of poliomyelitis, particularly those resulting from tonsillectomy.

Von Magnus and Melnick[4] demonstrated an increased susceptibility to experimental poliomyelitis in tonsillectomized Cynomolgus monkeys subsequently fed virus. Our study was designed to determine if human gamma globulin will modify or prevent paralytic poliomyelitis in tonsillectomized Cynomolgus monkeys, and if the administration of gamma globulin will interfere with the development of active immunity as determined by neutralization tests.

MATERIALS AND METHODS.

Two experiments were carried out on a total of sixty-six Cynomolgus monkeys, averaging in weight from five to six pounds. Experiment 1 involved the use of twenty-two animals and in Experiment 2, forty-four animals were used. Both experiments were carried out identically except that 1.0 ml. of gamma globulin per pound was injected intramuscularly in Experiment 1, 2.0 ml. of gamma globulin was employed in Experiment 2. The gamma globulin, however, was administered in two doses in Experiment 2, 1.0 ml. per pound just prior to feeding the virus and 1.0 ml. per pound three days later. Only twenty-four animals were tonsillectomized in Experiment 2, twelve of which received globulin. Twenty non-tonsillectomized animals served as controls. All monkeys were tonsillectomized by the snare method following the use of ether anesthesia.

Eight hours after tonsillectomy, each animal was fed daily for seven days, bread bearing approximately 100PD$_{50}$ intracerebral doses of the Y-SK strain of poliomyelitis virus. The twenty control non-tonsillectomized monkeys were similarly fed. From 5.0 ml. to 10.0 ml. of blood were obtained for neutralization tests from the femoral vein of all animals prior to exposure to the virus and during convalescence. Daily observations for signs and symptoms of illness were made.

Animals which died were autopsied immediately, and others showing evidence of illness were sacrificed at the termination of the experiment. In Experiment 2 only the animals which died or were ill were sacrificed. The cervical and lumbar enlargements of the spinal cord were sectioned and examined microscopically. The criterion for poliomyelitis was the demonstration of characteristic pathologic lesions in the cord, such as round-cell infiltration of the perivascular and interstitial tissue, destruction of neurons and neuronophagia.

White mice, in groups of eight, were used in carrying out standard neutralization tests. The sera obtained prior to feeding monkeys virus were pooled, whereas the convalescent sera were tested separately. The technique used was essentially as outlined by Paul and Riordan[5] except that the virus was incubated with monkey serum for one hour (37° C.) then refrigerated over night prior to the intracerebral inoculations. The virus suspension was titrated in mice and the LD_{50} dose was found to be $10^{-3.4}$. All mice were inoculated with 50 LD_{50} doses in the neutralization tests.

RESULTS.

Experiment 1: Six of the eleven tonsillectomized monkeys fed poliomyelitis virus and treated with 1.0 ml. of gamma globulin per pound remained normal. Two monkeys developed marked paralysis in twelve and seventeen days, respectively. Two other animals had fever and questionable symptoms of the disease. The remaining monkey, the smallest in the group, died on the third day after operation without showing clinical evidence of poliomyelitis. At autopsy, no histologic lesions characteristic of the disease were observed in the cord. On the other hand, the two animals which had marked paralysis showed microscopic lesions typical of poliomyelitis. The examination of the spinal cord from the other monkeys revealed no evidence of the disease.

Among the eleven tonsillectomized monkeys fed virus and not treated with gamma globulin, two developed the bulbar form of poliomyelitis, three showed moderate paralysis, and four animals exhibited *questionable* signs, including fever. Two *remained well*. Histologic examination of the cord, however, demonstrated typical lesions in only three cases.

Thus, unquestionable clinical and pathologic evidence of poliomyelitis was *observed* among two of the eleven tonsillec-

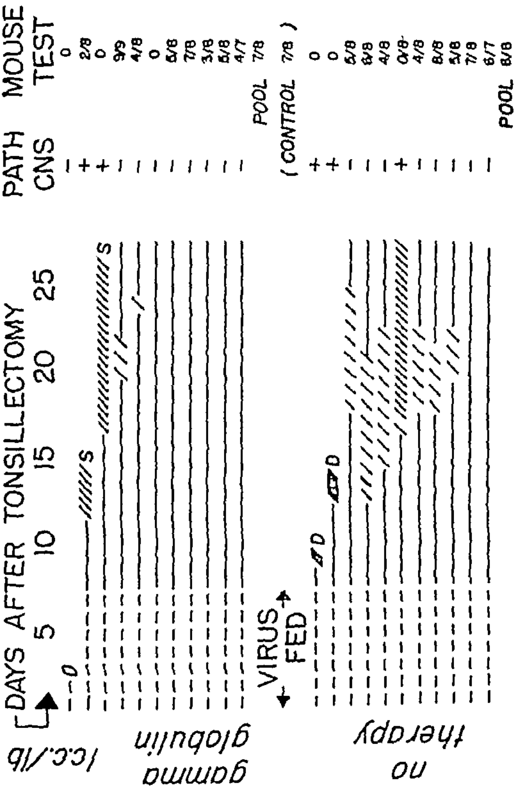

Fig. 1. This chart shows the clinical, pathological and serological results of Experiment 1; the intensity of involvement, illustrated by single slanting bars, indicates mild or questionable weakness, several bars indicate definite weakness or paralysis, and the solid bar represents signs and symptoms of bulbar disease. S means the animal was sacrificed and D indicates that the animal died.

tomized monkeys in the group injected with gamma globulin and in three of the tonsillectomized monkeys which did not receive gamma globulin; therefore, no significant difference was detected among the two groups. The course of the experimental infection in the two groups is graphically illustrated in Figure 1. The average fever record of these two groups of animals is shown in Figure 2.

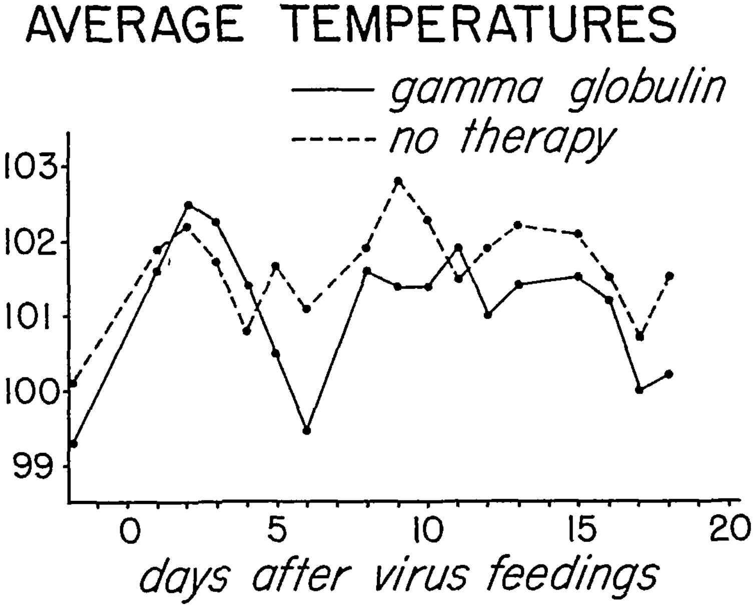

Fig. 2. This chart shows the average daily rectal temperatures in the two groups of animals in Experiment 1.

Experiment 2: Four of twelve tonsillectomized monkeys fed poliomyelitis virus and treated with 2.0 ml. of gamma globulin per pound presented symptoms suggestive of the disease. In the cords of only two of these was it possible to demonstrate typical microscopic lesions. The eight other animals remained normal and were not sacrificed.

Five of the twelve tonsillectomized monkeys in the group that were fed virus and received no gamma globulin developed symptoms suggestive of poliomyelitis, but the cords of only two of six that were sacrificed and examined exhibited pathologic lesions characteristic of the infection. (See Figure 3.) Thus, only two in each group of the treated and untreated were proved to have *poliomyelitis.*

None of the twenty untreated, non-tonsillectomized monkeys

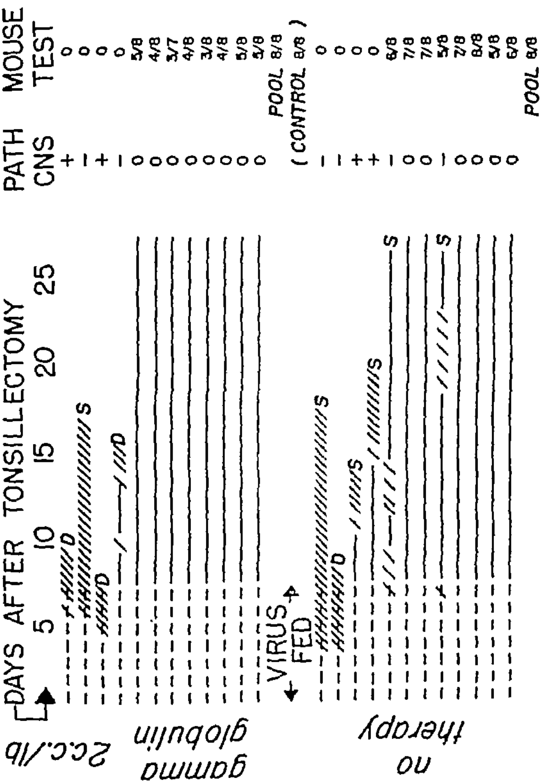

Fig. 1. This chart shows the clinical, pathological and serological results of Experiment 2; the single slanting bars indicate mild or questionable weakness, several slanting bars show definite weakness or paralysis; S means the animal was sacrificed and D indicates the day of death of the monkey.

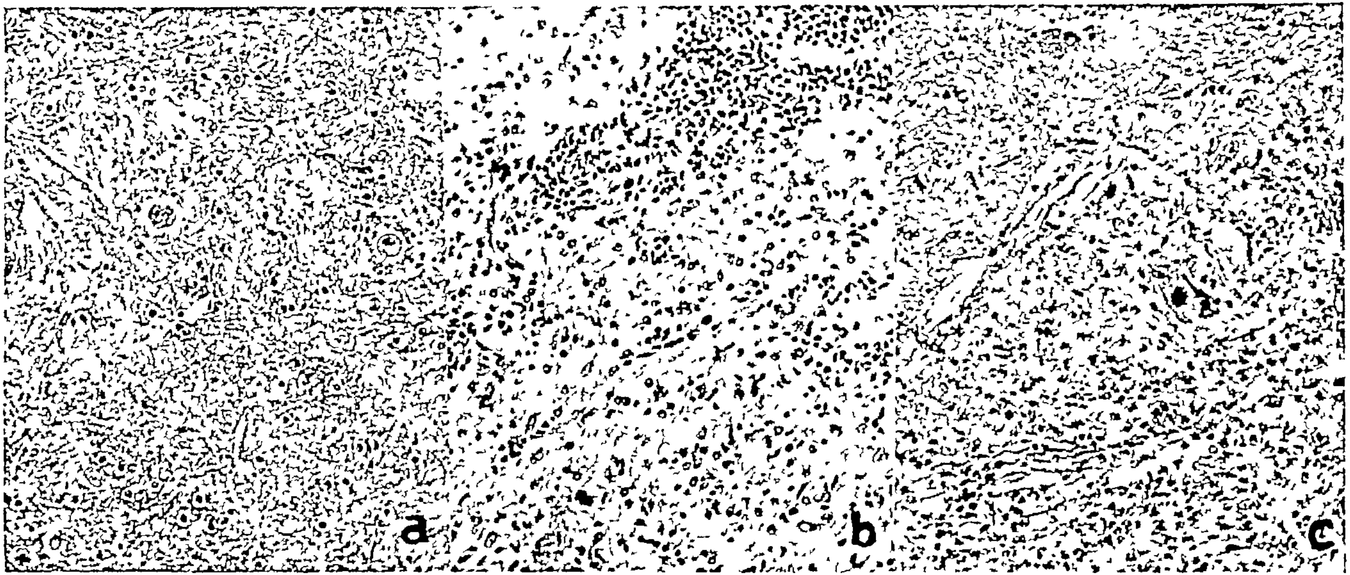

Fig. 4. (a) Photomicrograph of a spinal cord of a normal monkey, (b) and (c) Photomicrographs of the spinal cord lesions of monkeys which died of experimental poliomyelitis.

fed poliomyelitis virus developed typical symptoms of the disease. One had temporary weakness of a single extremity and was sacrificed together with six others from the group. No evidence of infection with poliomyelitis was detected in their spinal cords.

The characteristic spinal cord lesion of experimental poliomyelitis in monkeys consists of perivascular and interstitial round-cell infiltration with neuronophagia and neuron destruction. These changes are illustrated in the accompanying Figure 4.

The pooled sera from the normal monkeys showed no evidence of protection of mice against the Y-SK strain of poliomyelitis virus. On the other hand, the convalescent sera from the tonsillectomized, infected monkeys, including the treated and untreated groups, suggested a sparing effect. The serum from the non-tonsillectomized monkeys fed virus failed to protect mice.

The question of the devolpment of active immunity following the use of gamma globulin is partially answered by the results of the mouse neutralization tests on the convalescent blood samples of the eight animals in Experiment 2 which received 2 ml. of gamma globulin. At the 21 day period, these tests read as follows: 4/8, 2/8, 1/7, 2/8, 2/8, 4/8, 3/8, and 2/8. At the 30 day period the same tests read as follows: 5/8, 4/8, 3/7, 4/8, 3/8, 4/8, 5/8, and 5/8. The pool of serum on these animals prior to infection was 7/8 at 21 days and 8/8 at 30 days.

Gamma Globulin Dilution	Results After 3 Weeks
1:10	1/8*
1:20	1/8
1:40	1/8
1:80	1/8
1:160	1/8
1:320	2/8
Control	6/8

*The numerator designates the number of animals that died during the observation period.

The titration of the gamma globulin in mice for neutralizing antibodies against the Y-SK strain of poliomyelitis virus indicates a satisfactory titer by the mouse neutralization method in the gamma globulin used in these experiments.

The Y-SK strain used in these experiments is a Lansing type of virus, being a homotypic strain.

DISCUSSION.

The results of these experiments point out that either one or two ml. per pound of body weight of human gamma globulin intramuscularly failed to modify or prevent paralytic poliomyelitis in Cynomolgus monkeys. Although the dosage may be considered large, it is evident that larger doses will be required to demonstrate a significant sparing effect. The amount of virus required to produce the paralytic form of the disease by oral feeding may be an important factor in this result. The gamma globulin might have shown evidence of protection if smaller doses of virus had been introduced intracerebrally.

Failure to induce experimental poliomyelitis in twenty control non-tonsillectomized animals by the same oral feedings of virus indicates a significant difference between this group and the four other groups of tonsillectomized monkeys which include forty-five animals. Nine animals in these latter four groups, or twenty per cent, developed proven poliomyelitis. The mouse neutralization tests which were almost completely negative in the control group fed virus but not tonsillectomized would indicate that we failed to produce the disease in any form in these animals.

Von Magnus and Melnick[4] showed thirteen of fourteen (93 per cent) monkeys developed poliomyelitis after having been fed virus following tonsillectomy. In their control animals, two of fifteen (13 per cent) were positive. In a pharyngeal spraying experiment, Sabin[6] failed to infect eight Rhesus monkeys after tonsillectomy, but he did show that when inoculated in the tonsillopharyngeal region poliomyelitis developed with greater frequency than when inoculated intra-abdominally, intracutaneously, or subcutaneously. Several works[7,8,9] failed in various experimental studies, involving tonsillar areas, to produce disease in Rhesus monkeys; but Burnet, Jackson, and Robertson[10] infected nine of nineteen Cynomolgus monkeys with swabbing experiments.

Recently, Rhodes and Clark[11] reported that both placental and plasma gamma globulins contain Lansing antibody and that in mice the 50 per cent neutralizing titer was $10^{-3.5}$. They also showed a significant sparing effect when inoculated in *mice 24 hours before a cerebral challenge of Lansing virus.*

It does not seem necessary to review the controversial tonsil-poliomyelitis problem which has been summarized in several recent reports.[1,12]

Aycock[1] *has pointed out that there are many other possible* contributing factors as important as tonsillectomy in increasing the susceptibility to poliomyelitis both as to frequency and severity. Some of these factors might be briefly mentioned as reports in the literature[1] [illegible] indicate and include pregnancy, over-exertion, inoculations for active immunization, and operations each playing a significant rôle in the increased susceptibility to the paralytic forms of poliomyelitis.

Parenthetically, it should be reemphasized that poliomyelitis is a widespread disease, for the most part benign and trivial, often being subclinical and that the degree of severity is determined, not so much by the circumstances of exposure to the virus, but by factors which make for increased susceptibility in a certain few of the many who are exposed. When *faced with a decision in this matter, it should be borne in* mind that we are unaware of the circumstances of exposure and should do all we can to prevent or delay any procedure which might increase the susceptibility of our patient.

SUMMARY AND CONCLUSIONS.

1. Concentrated human gamma globulin was used in an attempt to modify or prevent experimental poliomyelitis in tonsillectomized Cynomolgus monkeys. In two groups of monkeys in which doses of 1.0 ml. and 2.0 ml. of gamma globulin per pound were administered, no significant differences were observed in the treated and untreated animals.

2. Mouse neutralization tests were performed on forty-six of the surviving animals. The tests failed to show evidence of poliomyelitis antibodies in thirty day specimens from the non-tonsillectomized controls; however, these tests do show some evidence of antibody production in animals that were tonsillectomized and given gamma globulin.

3. Incidental to these observations, it was determined that an attack rate of twenty per cent (nine of forty-five monkeys) occurred in the tonsillectomized animals, whereas in the non-operated control group of twenty monkeys fed the same virus in an identical dosage, no poliomyelitis developed.

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PAPILLOMA OF THE LARYNX WITH EXTENSIVE LUNG INVOLVEMENT.

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The propensity of papilloma of the larynx in children to recur and proliferate after removal is a source of great interest and considerable chagrin to the otolaryngologist. The slightest trauma, even sponging with gauze, seems to promote the spread of these growths. Most of the extensions are above the glottis along the lines of instrumentation, while the relatively infrequent subglottic extensions occur mostly along the tracheal stoma in patients wearing a tube. There are cases on record, however, of more diffuse disseminations from primary laryngeal lesions,[1-5] and of papilloma of the trachea, with or without involvement of the larynx.[6-9]

Since 1922, with Ullmann's pioneer work in transplanting human laryngeal papilloma,[10] investigations have suggested that it is not a true neoplasm but is the result of a virus infection. This theory is supported by several features of Ullmann's original work and by subsequent observation by others of these and other papillary lesions. Ullmann succeeded in growing small, flat warts on the skin of his arm and on the mucosal surfaces of dogs by inoculation with papilloma removed from the larynx of a young boy; then, using the warts on his arm as donor areas, he transferred the lesions to the skin of a human volunteer. On this second transfer the incubation period was shortened by half and the virulence of the etiological agent increased to such a degree that the resulting lesion required surgical excision. Finally, using a bacteria-free infiltrate, he effected transfers in two of six attempted cases.

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Papillary lesions of other parts of the human body, such as venereal condylomata and plantar warts, occur in association with chronic inflammation. Jarvi[11] reported a papilloma of the maxillary sinus whose cells contained acidophilic intracytoplasmic inclusion bodies with mucus mantles, quite characteristic of a virus infection. Intranuclear bodies have been described in plantar warts, but these may represent products of degeneration. Some extremely interesting work in the production of papilloma in animals with recovery of the virus has been done.[12-14]

On the other hand, there appears to be a definite endocrine influence on the course of laryngeal papilloma. They tend to disappear spontaneously at puberty and have responded to local estrogen therapy by Broyles.[15] Holinger[16] reports an interesting case of papilloma of the larynx in a woman, which regressed during each of three pregnancies, then recurred with the onset of menstruation each time.

The following case is further evidence, in my mind, of a virus etiology of laryngeal papilloma. It is difficult to believe that so extensive a process could have been caused by anything but an infection.

A seven-year-old colored female came to the E. N. T. Clinic at Johns Hopkins Hospital, wearing a tracheotomy tube which had been in place for two and one-half years At four and one-half years of age, she had developed hoarseness which progressed to aphonia and dyspnea. Her family physician made a chest X-ray and blood test, both of which were reported normal. She was referred to an otolaryngologist who performed a tracheotomy and removed tissue from the larynx which proved to be squamous papilloma. He removed this tissue every three weeks for the next two and one-half years, at which time the family left the city.

On her first admission to our hospital she had a productive cough, which had been present from the time of the tracheotomy There was no history of any other childhood disease There were moist rales at both lung bases and fluoroscopy showed increased bronchovascular markings.

Laryngoscopy on admission showed extensive papillomatosis of the larynx and pharynx The glottic chink was completely occluded and the tissue proved to be squamous papilloma The patient's cough seemed to be worse when she was obliged to breathe entirely through the tracheotomy tube, and she expressed a desire to have the glottic chink kept clear enough to allow for exchange through the upper air passages with the tube completely corked. This was accomplished by several removals of the papilloma during her hospitalization, and the airway was quite adequate at the time of her discharge She returned to the E. N. T. and Pediatrics Dispensaries at one-month intervals and was laryngoscoped under general anesthesia at each visit so that an adequate airway was maintained by removal of small bits of tissue. The general condition of the larynx gradually improved. On one visit, seven months after initial admission, she was found to have a temperature elevation (99.4°) and an increase in the basal rales. A chest X-ray was made and it revealed the multiple cystic areas shown in Fig. 1. One month later, she developed right middle and right lower lobe pneumonia, which cleared up on sulfadiazine; she was then started on a daily maintenance dose of this drug Seven months later, on one of her regular visits to the Pediatrics Clinic, she complained of some increase of dyspnea; fluoroscopy showed a fluid level in the left chest, which was thought by the interne on duty to be empyema. His attempt to aspirate this fluid produced a sudden respiratory crisis with left-sided pneumothorax. She was given oxygen and an attempt was made to relieve the air pressure by a second thoracentesis, but this resulted in tracheal obstruction with cessation of respiration

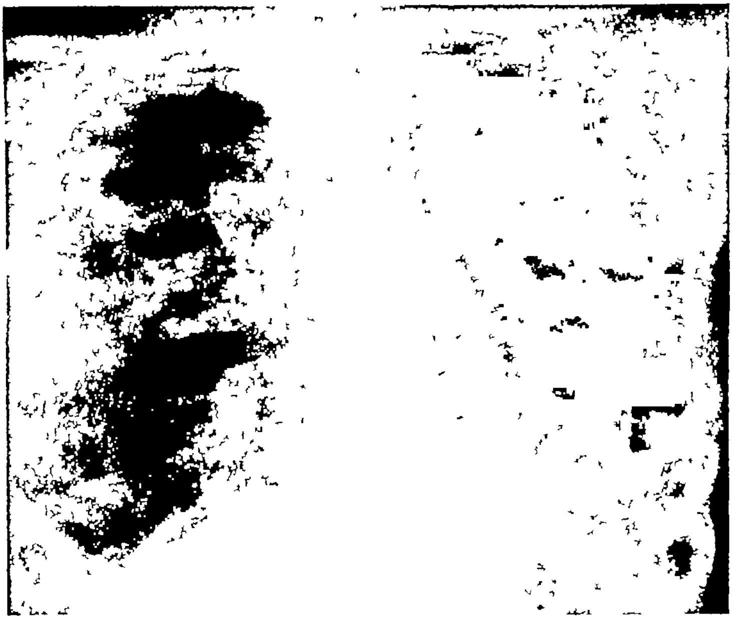

Fig 1 Multiple cystic areas in both lung fields Three years previously, at time of onset of hoarseness and dyspnea, lungs were reported normal by X-ray examination.

Autopsy revealed an almost unbelievably extensive involvement of the trachea, bronchi and lung by various sized nodules and cavities. It was hard to believe that this condition could have been compatible with life as long as it had. The huge nodular mass nearly filling the lower trachea was the immediate cause of the fatal tracheal obstruction. On gross examination, the solid masses appeared to be made up of the same tissue which filled the larynx. This was also true of the tissue lining the walls of the cavities. Microscopic examination verified this impression, revealing small peribronchiolar nodules made up of squamous cells (see Fig. 3), larger masses of squamous cells and large cavities lined by typical squamous papilloma (see Fig 4) identical with that in the larynx. Nowhere was there any morphological evidence of malignancy.

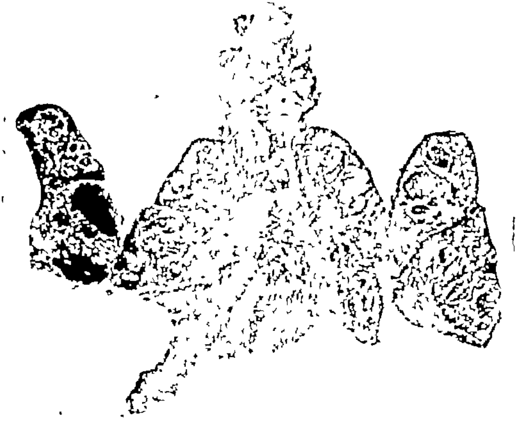

FIG. 2. Autopsy specimen of larynx and lungs showing masses of papilloma involving the larynx, base of tongue, trachea, bronchi and lung parenchyma. The large cavities are lined by tissue which is identical, both grossly and microscopically, with that in the larynx.

DISCUSSION

Since no involvement of the pulmonary lymphatics or blood vessels was reported in this case, I feel that two mechanisms were involved in the pathogenesis of these lesions: *1.* aspiration implantation, *2.* development *in situ*. By the former process, small particles becoming detached from the growth in the larynx, from time to time, could be aspirated out into the bronchial tree to a point distal to the effective action of the cough mechanism, where they could lodge, attack the bronchiolar epithelium by means of their virus laden tissue juice and produce an area of metaplasia which would develop

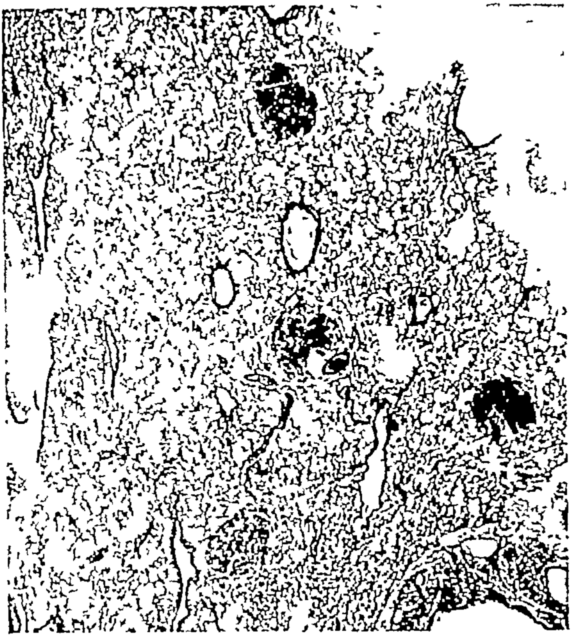

Fig. 3. Low-power view of the small peribronchiolar nodules. These are composed entirely of squamous cell aggregations identical with those in the larynx.

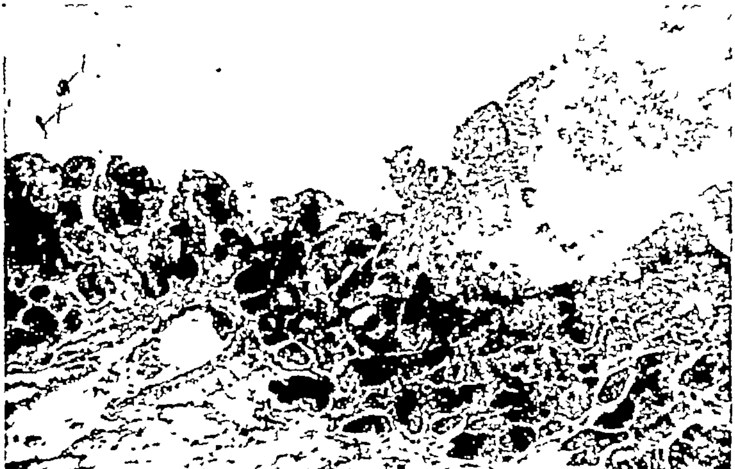

Fig. 4. Wall of a typical cavity showing masses of squamous cells. The sloughing area on the right illustrates the manner of formation of these cavities from solid masses of squamous cells.

into a nodule of squamous cells. This would account for the distally located peribronchiolar nodules since larger particles would not by-pass the bronchi and would easily be coughed out. Fig. 5 shows a bronchiole whose wall has become distorted and replaced by masses of squamous epithelium. Its lumen shows columnar ciliated epithelium around half the circumference, the other half being a low cuboidal ciliated epithelium continuous with the squamous epithelial mass making up one of the nodules The lumen of this bronchiole if followed further proximally and distally would probably show a point where this particular nodule started to grow, probably a tiny particle of tumor which had been aspirated from the larynx. Although we were not able to demonstrate a plug of papilloma lying free in a small bronchus, Hitz and Oesterlin[8] reported a case in which a small bronchus was occluded in this manner, and in which nodules of squamous cells were found in the parenchyma. Our case, however, showed one striking difference from the one they reported, in that they found no tumor in the lower half of the trachea or larger bronchi. It is the presence of the latter lesions that makes me believe that a

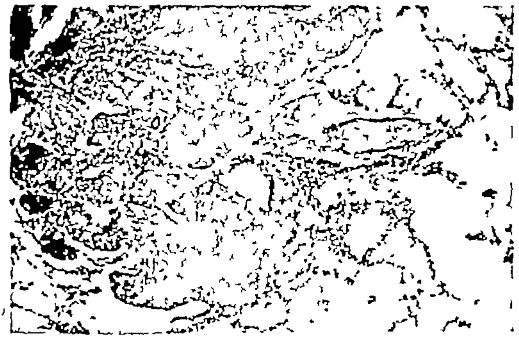

Fig 5 Mass of papilloma growing from wall of bronchiole. This particular nodule probably developed from a fragment aspirated from the larynx, initiating the process proximal or distal to the plane of this section.

second mechanism, namely, development *in situ,* was operative in this case. This may have been a generalized laryngotracheobronchitis of virus origin, with the laryngeal and pulmonary lesions developing concomitantly; however, the normal chest film prior to tracheotomy; the fact that the cough had not developed until after the tracheotomy, and the failure of fluoroscopy on hospital admission two and one-half years later to show anything more than prominent bronchovascular markings, lends weight to the impression that the lung lesions developed after those in the larynx. A more likely explanation seems to be that the tissue juices being released from the papillary growths in the larynx by trauma from cough or instrumentation could easily invade the tracheobronchial system by aspiration and attack certain portions of the epithelial lining, eliciting a response characterized by the formation of papilloma. After the masses of papilloma attained a certain size, the central portions became necrotic and sloughed out as shown in Fig. 4, leaving a cavity. Intervening stages of this process are suggested by Fig. 6.

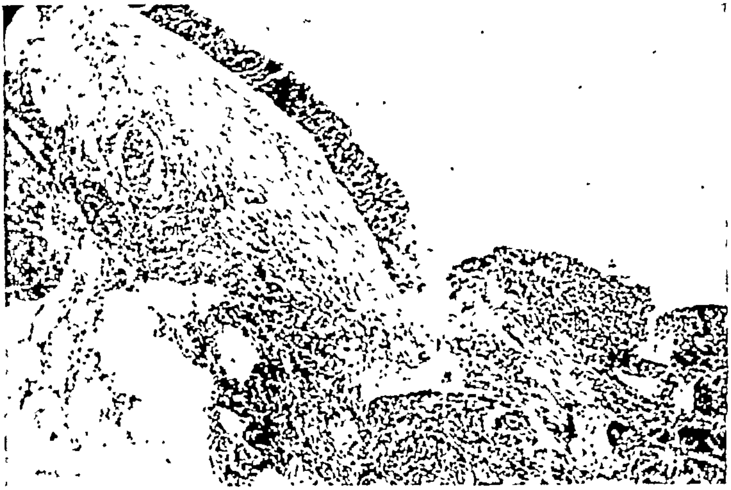

Fig. 6. Area illustrating transition from metaplastic respiratory epithelium to papilloma.

As a concluding suggestion, we should be aware of the possibility of lung involvement in every case of childhood papilloma of the larynx we see. Pulmonary extensions of a lesser degree than that reported above may be more common than we realize since this disease is usually self-limited and can seldom be investigated as thoroughly as was this case.

I express my appreciation to Dr S J. Crowe and Dr. E. N. Broyles for allowing me to report this case which I saw during my residency at Johns Hopkins Hospital.

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A CASE OF RECEDING CHIN AND HUMP NOSE WITH CORRECTIVE METHOD.*

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The combination of a receding chin and a prominent hump nose gives a facial contour sometimes known as bird-face, which may be very deforming when either is present even in a moderate degree.

This irregularity of the facial features is usually congenital and developmental. The exact cause of the condition is not entirely clear, though in some cases there is a strong hereditary influence as seen in similar features throughout the family. Experimental study on animals, on the other hand, has demonstrated that growth of the mandible can be varied by the administration of estrogens. This result is manifested as an arrest of growth in the anteroposterior direction. It is well known that changes take place in bone growth in acromegaly where there is disturbance of hypophysical function.

The mandible, like most of the other bones of the skull, has a membranous rather than a cartilaginous origin. The only cartilage that enters into the early structure of the mandible is known as Meckel's cartilage, which is derived from the first branchial arch of the embryo. This narrow strip of cartilage is surrounded by embryonic mesenchymal membrane and extends the entire length of the part which becomes the lower jaw. The proximal part of Meckel's cartilage gives rise to the incus and the malleus of the middle ear.

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About the thirty-ninth embryonic day (15 mm.), centers of ossification can be distinguished in the mandibular tissue. Bony growth goes on to enclose the anterior two-thirds of Meckel's cartilage, while that portion of the cartilage that is associated with the ramus does not become a part of it but undergoes fibrosis and finally forms the sphenomandibular ligament. At the distal end, or the symphysis, the two halves are at first united by fibrous tissue, which ossifies later.

The appearance of the small chin may be due either to limitation of growth of the anterior extremity of the mandible, or to lack of development in the posterior third of the body. As a rule, one or two small bony eminences develop on the anterior surface of the chin itself, and when these are absent there is a somewhat receding chin line; but more often, this chin contour results from lack of full development, either at the angle of the ramus or in the neck of the condyle.

The profile line of the chin depends essentially on the angle of the ramus. Dentition notably alters the size of the angle present at birth, and growth at this time tends toward a marked change in the mandible. After the eruption of the first teeth, the angle of the ramus with the body of the mandible is about 140 degrees, but after mature growth this becomes more nearly a right angle.

The unesthetic effect of the combined prominent hump nose and the receding chin is very evident. The profile line is markedly altered to appear in many instances as a right angle at the nasal tip, and in extreme cases it may be more acutely angled. With this abnormality in the case presented here there is very evident facial asymmetry, involving also the detail of the external nose. There is slight lowering of the left corner of the mouth, and a degree of ptosis of the right eyelid, together with very slight internal strabismus.

The history of the case reported is that of a young girl, 15 years of age, said to be somewhat slow in her mental reactions but already in high school and apparently doing passing work, although this is difficult to determine when pupils are advanced according to age rather than according to the measure of their actual ability. The birth was normal, not difficult, nor instrumental. She was a rhachitic baby and as a young child was always considered "frail"; she had measles at five years of age, followed by an attack of infantile paralysis at eight. The left side was affected during the acute attack but with no resulting signs at the present time.

The girl did not wish to have the corrective operation, although she was conscious of being unpleasantly conspicuous in appearance, and of the habit of the neighboring children calling her taunting names and jeering at her on account of her facial characteristics. She was careless of her personal appearance, not dressing neatly or becomingly, and preferred to stay by herself as much as possible, rather than share any social life. The mother of the girl sought the operation for her daughter and was insistent on having it done, since she was greatly embarrassed when in public with the daughter. People stared at them, and the mother felt that she, too, was being under criticism; that the girl's appearance was a reflection upon herself. This was apparently the motivating idea in requesting that the plastic operation be carried out.

To determine just what changes were necessary to secure a suitable esthetic facial contour, a plaster mask was made, upon which the desired designs were sculptured, and it was used in performing the actual operation.

Although this is not customary, the procedure of removing the hump and installing the chin implant was carried out under general anesthesia, since the patient was inclined to be uncooperative, making a local anesthetic alone inadvisable. The entire correction was done as a single stage operation.

With the patient under the general anesthetic, for which ether was used, the external nose was infiltrated in the usual manner with novocaine (procaine) and adrenalin to insure hemostasis. The primary intranasal incision was made on the left side with a Bard-Parker No. 11 blade, cutting along the intranasal ridge from its outer extremity toward the septum, and taking particular care not to puncture the skin as the knife advanced upward between the lower and upper cartilages. When the knife was withdrawn, a small gauze sponge was inserted to check bleeding, and this *first step* was repeated on the right side. A Joseph double-edged knife was then used on the left, through the incision already made, to undermine the skin over the nasal dorsum, and repeated on the right side. The periosteum of the nasal bones was next elevated to make a small pocket using a McKenty sharp elevator, followed by insertion of a periosteal elevator, with which the periosteum over the entire hump was separated from the underlying bone. When the right side had been

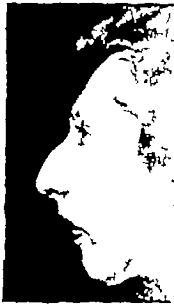

Fig 1

Fig 2

treated in a similar manner the elevation of the periosteum over the bony dorsum was complete. A button-end knife was then inserted into the incision and extended upward under the periosteum to the upper border of the nasal bones and the blade then carried downward over the entire dorsum until its tip appeared in the incision on the right, from which point it was continued further downward, nearly to the septal tip where the direction was changed to follow the lower border of the septum, until the anterior nasal spine was reached.

For removal of the bony hump, right and left bayonet saws were used, one on each side, so placed as to separate the hump at the predetermined level, in order to secure an esthetic facial contour. The separated hump bone was removed through the incision with hemostatic forceps, and the remaining edges smoothed with a rasp, followed by trimming off the dorsum of the septal cartilage to follow the profile of the new line of the bridge.

The familiar rule that narrowing the bridge is always necessary on removal of the hump was followed. This step was accomplished by sawing through the anterior maxillary processes on both sides and pressing both bone fragments into position with the thumb and finger.

In addition to narrowing the hump, the nose had to be shortened and the tip suitably sculptured. This required removing a triangular section from the lower border of the septum and excising a narrow strip along the lower border of the upper lateral cartilage, which was done with curved scissors.

To narrow the tip, a segment of the angle of the lower lateral cartilage was freed by making a circular incision just inside the vestibule and undermining the skin both on the external and vestibular sides of the cartilage. The amount of cartilage removed from the angle in this step determines the final width of the reconstructed nasal tip.

After all of the parts were molded into the desired position, a dressing of two strips of adhesive was applied, one over the dorsal surface of the tip and the other over the lower nasal surface, with the ends of the two bands crossing at right angles on either side of the nose. Over this adhesive strapping, a cast of dental mold compound was fitted and lined with a pad of soft cloth to prevent skin irritation. The cast was finally strapped on with narrow strips of adhesive, placed across its upper part and at its lower extremity, from cheek to cheek.

For correcting a receding chin a number of different surgical methods have been used, the choice depending largely upon the degree of the distortion and upon the angle of the ramus. In this case, no bone surgery was used, but the lack of prominence of the chin line was filled in by insertion of the bony hump tissue which was removed from the nasal dorsum. Preserved cartilage, minced cancellous bone and a number of other materials have been used as implants in such cases.

The operation was begun with a Bard-Parker No. 15 blade, by making an incision of about one-fourth inch in length in the slight fold that appears just beneath the tip of the chin, when the head is flexed on the chest. More often, it is the custom to make the incision for this operation in the depression on the anterior surface of the chin, just below the lower lip. Through this small incision a bed was made for the graft by undermining the periosteum to form a pocket of the required size. The graft was then inserted and carefully manipulated manually so that it lay on a position over the front of the chin (not over the tip) to make an esthetic curve, and so improve the profile contour. The skin was sutured and a simple dressing applied.

Such cosmetic operations are nearly always concerned with an emotional factor. This young girl now appears more like her school companions, and instead of feeling as though she were a curiosity, and preferring to avoid others, and neglecting her personal appearance, she now is quite particular about her toilet and dress, goes to the hairdresser and uses facial cosmetics, with the result that her whole social life is changed by being included instead of being shunned. Her mother is greatly pleased to find that there has been such a satisfactory result following the corrective operation.

This case furnishes one more testimonial to the saying that "every human being has a right to look human."

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HONORARY DEGREE TO DR. JULIUS LEMPERT.

The first honorary degree of Doctor of Medicine to be bestowed upon an otologist by the venerable Karolinska Mediko-Kurugiska Instituet (Caroline Medical Surgical Institute), of Stockholm, Sweden, was given to Dr. Julius Lempert, of New York, on May 30, 1951, for his contributions to otology.

The awarding of degrees took place in the Stockholm Town Hall, one of Europe's most beautiful architectural structures. In strict dignity and formality, Prof. Arvid Wallgren officiated at the presentation ceremony. The degree was awarded to Dr. Lempert in appreciation and admiration by the faculty of this renowned institution for his surgical contributions and for his teaching of others in the advancement of otologic surgery, particularly to those members of that department of the Caroline Institute.

Dr. Lempert's friends throughout the world are delighted to learn that he has received one of the most distinguished honors in medicine.

AMERICAN COLLEGE OF SURGEONS PROGRAM FOR 1951 SAN FRANCISCO CLINICAL CONGRESS, MARK HOPKINS HOTEL.

Otolaryngology Section.

AFTERNOON SESSIONS

Tuesday, November 6, 3:30 to 5:00 p.m.

Symposium on "TREATMENT OF LARYNGEAL PARALYSIS"
"INTRALARYNGEAL APPROACH."
William C. Thornell, M D , F A C S , Cincinnati.

"EXTERNAL OPERATION."
DeGraaf Woodman, M.D , F.A C.S , New York.

"MANAGEMENT OF CICATRICIAL STENOSIS OF LARYNX."
Robert C. McNaught, M D San Francisco.

SUMMARY
By the Chairman.

Wednesday, November 7, 3:30 to 6 00 p.m.

"CLINICAL EVALUATION OF DIZZINESS"
David D. DeWeese, M D , Portland, Oregon

Discussors:
George L. Pattee, M D Denver
Albert J. Lubin, M D San Francisco

"THE NERVE-DEAF CHILD"
Victor Goodhill, M.D., F A C S Los Angeles.

Discussors:
Paul J. Moses, M.D San Francisco
Mrs. Spencer Tracy, Los Angeles

Thursday, November 8, 1:30 to 3:00 p.m.

Symposium on "MANAGEMENT OF FACIAL PARALYSIS."
"BELL'S PALSY."
Robert C. Martin, M D., San Francisco

"TRAUMATIC LESIONS OF THE FACIAL NERVE."
Frank D Lathrop, M.D., F.A C.S , Boston.

SUMMARY
Sterling Bunnell, M.D., San Francisco.

EVENING SESSIONS

Tuesday, November 6, 8:30-10:30 p.m.

Symposium on "HEAD AND FACE PAIN.'
"PAIN OF SINUS ORIGIN."
Harold Owens, M.D., F.A.C.S., Los Angeles.

"PAIN DUE TO AUTONOMIC DYSFUNCTION."
Neill F. Goltz, M.D., St. Paul.

"PAIN DUE TO INTRACRANIAL DISEASE."
Howard C. Naffziger, M.D., F.A.C.S., San Francisco.

"PSYCHOSOMATIC ASPECTS OF HEAD AND NECK DISCOMFORT."
Knox H. Finley, M.D., San Francisco.

Wednesday, November 7, 8:30-10:30 p.m.

Symposium on "FACIAL INJURIES."
"FRACTURES OF THE NOSE."
Jay Gilbert Roberts, M.D., F.A.C.S., Pomona, California.

"FRACTURES OF MAXILLA."
Claire L. Straith, M.D., F.A.C.S., Detroit.

"FRACTURES OF MANDIBLE."
Reed O. Dingman, M.D., F.A.C.S., Ann Arbor.

"ASSOCIATED FACIAL LACERATION."
William C. Huffman, M.D., F.A.C.S., Iowa City.

Thursday, November 8, 8:30-10:30 p.m.

Symposium on "CARCINOMA OF THE LARYNX."
"CARCINOMA OF THE LARYNGO-PHARYNX."
Lewis F. Morrison, M.D., San Francisco.

"CARCINOMA OF LARYNX WITH CERVICAL METASTASES."
Walter P. Work, M.D., San Francisco.

"X-RAY THERAPY."
L. Henry Garland, M.D., San Francisco.

SUMMARY
Louis H. Clerf, M.D., F.A.C.S., Philadelphia.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

September 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., [illegible] W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, [illegible] Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H Maico; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303; Micronic Star Model. (See Silver Micronic.)

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-1 (Whisperwate).

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model J (Tiny-Myte); Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.

Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)

Manufacturer: Micronic Corp, 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co, 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.

Manufacturer: Sears-Roebuck Co, 925 S Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.

Manufacturer: Micronic Co, 727 Atlantic Ave., Boston 11, Mass.
Distributor: Sears-Roebuck Co, 925 S Homan Ave, Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnston Mfg. Co, 703W. 40th St., Minneapolis, Minn.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp, Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer: Sonotone Corp Elmsford N Y

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc, 2454 S. Michigan Ave, Chicago, Ill.

Televox Model E.

Manufacturer: Televox Mfg Co, 117 S Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.

Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube Model No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Council Meeting: Waldorf-Astoria, New York City, Jan. 12, 1952.
Southern Section: Academy of Medicine, Atlanta, Ga., Jan. 14, 1952.
Middle Section: Indianapolis Athletic Club, Indianapolis, Ind., Jan. 28, 1952.
Western Section: County Medical Bldg., Los Angeles, Calif., Jan. 19,20, 1952.

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Meeting: Palmer House, Chicago, Ill., Saturday, Oct. 13, 1951.

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PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

Meeting. Third Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Havana, Cuba, January, 1952. Dr. Jose Gros. For information write Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

SECOND LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

Time and Place: Sao Paulo, Brazil, July, 1951.
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Time: 6.00 P.M., fourth Monday of each month from September to May, inclusive.

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Trèsoreo: Dr. Mario Arenas A.

THE
LARYNGOSCOPE.

VOL. LXI NOVEMBER, 1951. No. 11

A REVIEW OF THE AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1950.*

FRANCIS LEJEUNE, M.D., and MILES LEWIS, M.D.,
New Orleans, La.

ANATOMY.

The presence of a canal extending from the depth of the sella turcica to a point just behind the rostrum of the sphenoid has been reported many times and has been named the craniopharyngeal canal. Its presence is frequently noted in the higher apes. It is not uncommon in infancy and is rather rare in adults. Heretofore it has been assumed to be a persistent opening from the growth of Rathke's pouch. The investigation of Arey[1] disproves that origin and states that the craniopharyngeal canal is a channel formed during osteogenesis by the growth of blood vessels into the bone marrow in this region in the fetus.

Adhesions of the nasopharynx consisting of string-like fibrous bands to actual sheets of scar tissue are commonly seen in routine examination of this area. Lion[2] describes a peculiar type of headache located around and behind the eye, which he attributes to inflammation and retention of material in the nasopharynx behind these folds. Various folds and adhesions are classified by the direction they take. Lion be-

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lieves these adhesions are caused by inflammatory change in this area and to a lesser degree by improperly performed adenoidectomies, and that only a small percentage are congenital. Treatment consists of early and properly performed adenoidectomy as a preventive measure and simple clipping of the fibrous bands once they are formed.

In a symposium on irradiation of lymphoid tissue in the nasopharynx, Schenck[3] discusses the anatomy, physiology and pathology of nasopharyngeal lymphoid tissue. He reminds us that at the chief portals of entry to the body there is urgent need of a mechanism of removing and attenuating organisms and of developing immunity against them. It is here that lymph cells in large numbers are found. Some of the masses of lymph cells are so large they have been dignified by the term "tonsil." The innumerable smaller collections of lymph cells, commonly referred to as "follicles" or "nodules," are scattered throughout the pharynx and adjoining structures. These lymph structures are an integral part of a universally distributed system made up of lymph channels and vessels, and of accumulations of lymphocytes called lymph nodes, of which the body contains from 500 to 1,000, representing 1 per cent of total body weight. Lymph tissue, whenever found, is composed of two chief constituents: the supporting connective tissue reticulum and the lymph cells contained within the meshes of the reticulum. Lymph nodules and lymph nodes are discussed in a most excellent manner. Lymph tissue and lymphocytes play an important rôle in resistance and are probably the site of antibody formation. Because of the clinical importance of excessive growth of lymph tissue in the nasopharynx, Schenck thinks it advisable that consideration be given the factors which govern the growth of lymph tissue in general. He enumerates the most important as diet, endocrine secretions, stimuli other than hormones, infection and heredity. Each of these is discussed at length. This article constitutes probably the most concise description of lymph nodules, nodes and tissue structure of the nasopharynx available and describes in an interesting manner the complicated function of the lymphatic system.

sisting of glossitis, black tongue, stomatitis, pharyngitis and monilia albicans infection, developed in 12, or 6 per cent. Bitterness, dry mouth and soreness of the mouth usually appear four to six days after administration of the drug. The more severe complications were seen in elderly or debilitated patients. The lesions usually subside in the week following discontinuance of the drug. Administration of vitamins did not alter the complications; patients receiving additional antibiotics had more severe complications. The opinion is expressed that the monilia is held in check by the bacterial flora of the mouth and pharynx and this balance is upset by the antibiotic.

Webb[9] reports a case of Guillain-Barré syndrome with respiratory and pharyngeal paralysis. With the aid of a respirator and tracheotomy the patient recovered and was able to walk after six weeks. This condition is a polyneuritis of unknown etiology with a good prognosis. Of 115 cases in the literature, 25 patients died, practically all of them of respiratory paralysis. Early tracheotomy and early use of a respirator would seem to prevent most of these deaths.

Smith[10] reports a case of tuberculous retropharyngeal abscess in a woman 35 years old, in whom cure was effected by surgical treatment and administration of streptomycin. The unusual feature of the case was the lack of involvement of the cervical vertebrae, as is usually the case in tuberculous abscesses in the pharynx.

Bryant[11] reports a case of retropharyngeal abscess in a boy six years old, which was thought to be secondary to infection in a regrowth of adenoid tissue. The abscess was successfully incised and drained, and two months later a mass of adenoid tissue was removed.

Schenck[12] presents a practical textbook type of discussion of several acute infectious conditions of the pharynx, and although it is fairly elemental, there are a few good points emphasized under each heading. Septic sore throat is due to hemolytic streptococcus; warm saline irrigations, local applications of tincture of benzoin and penicillin parenterally are recommended. Acute lacunar tonsillitis may be treated by applications of 25 to 50 per cent silver nitrate directly to the tonsils, using a small amount of the solution, warm saline irrigations and parenteral penicillin. It is important to study the blood picture carefully in patients with Vincent's angina to rule out leucemia. Treatment of Vincent's angina consists of applications of sodium perborate paste, parenteral and local administration of penicillin, and in some cases intramuscular injection of sodium cacodylate. When diphtheria is suspected, culture on Loeffler's medium should be done. Toxoid injections are imperative and penicillin is useful in combatting secondary infection. Peritonsillar abscess may be aborted by penicillin or sulfonamide therapy, but once fluctuation occurs, incision and drainage must be done. Acute pharyngitis may be treated by local application of compound tincture of benzoin and saline irrigations; it often heralds a systemic disease. Thornwaldt's disease, due to infection and cyst formation of a pharyngeal bursa, is treated by surgical removal of the bursa. Retropharyngeal abscess occurs almost entirely in children and is due to suppuration of the retropharyngeal lymph nodes; incision and drainage are employed once fluctuation occurs. Penicillin parenterally will usually prevent this serious condition.

Asherson[11] presents some cases with excellent radiographs to draw attention to the syndrome of achalasia of the cricopharyngeus. This condition, meaning failure of relaxation, may occur at the cardia or lower end of the esophagus, or at the upper sphincter of the esophagus, the cricopharyngeal sphincter. In the latter case it results from failure of this sphincter to relax when a bolus of food approaches it. Achalasia of the cricopharyngeus presents a typical radiological picture; on lateral Roentgenogram the hypopharynx is elongated and the valleculae are widened. Barium may take over five minutes to pass. This condition may be produced by pharyngeal paralysis such as caused by poliomyelitis, thyrotoxicosis, myopathy, unilateral recurrent laryngeal nerve paralysis, after partial pharyngectomy and in bulbar paralysis.

The condition is manifested clinically only by slight dysphagia, and on examination pooling of mucus is seen in the pyriform fossa. Esophagoscopy is indicated to rule out postcricoid neoplasm or a pharyngoesophageal diverticulum. Other conditions to be considered in the differential diagnosis of achalasia of the cricopharyngeal sphincter are Plummer-Vinson syndrome, foreign body of the esophagus, idiopathic dysphagia in the female and cricopharyngeal spasm produced by overdistension.

Crowe and associates[14] present a brief review of the highlights of the literature on otorhinolaryngology for the period of 1938-1948. The use of antibiotics is discussed; a small section is devoted to carcinoma of the sinuses, nasopharynx, mouth, hypopharynx, larynx and esophagus. The importance of chronic irritation as a cause of cancer of the mouth and pharynx as presented by Hayes Martin is mentioned.

DIAGNOSIS.

Lindsay and Perlman[15] present an excellent discussion of tests of chronic Eustachian tube obstruction. From a practical standpoint they divide the tests into three groups according to the age of the patient. In group one is placed the preschool child. The earliest practical indication of chronic tubal obstruction in this group is the presence of fluid in the middle ear. When distortions and scarrings of the drum are present, middle ear changes of irreversible nature have usually occurred. Early diagnosis depends upon the ability of the examiner to recognize the signs of a middle ear completely filled with fluid as well as one showing fluid lines. In group two are placed young school children whose hearing tests are limited in value. Otoscopic appearance is the chief indication of chronic tubal obstruction. Toynbee's maneuver, if positive, indicates grossly adequate function. In the third group are placed older children and adults.

Tubal function is determined by otoscopic examination and specialized tests. Detailed description of method employed in the detection of tubal function should be of utmost value to the student of otology who is not versed in these procedures.

DIVERTICULA.

The majority of diverticula originate between the oblique fibres of the inferior constrictor and the cricopharyngeus and are found on the left side of the neck. They consist of an outer fibrous and inner mucosal layer. The etiology is in dispute, but because of the occurrence of six cases in one family, Dunhill[16] favors a congenital defect rather than neuromuscular dysfunction as the cause. The usual symptoms are dysphagia, inability to swallow and regurgitation of food; severe cough may occur. The diagnosis is made from the history and Roentgenographic demonstration of the pouch.

For many years the two-stage procedure was preferred because of the danger of mediastinitis, and diverticulopexy, in which the pouch was elevated and sutured with the sac higher than the neck, has been used, particularly in older patients. More recently removal of the diverticulum in one stage has been widely employed. This is the method advocated by Dunhill for those experienced in neck surgery. The pouch is examined endoscopically only in patients in whom the possibility of carcinomatous invasion is suspected. The technique described is the generally accepted one in this country.

Because he believed that the importance of a pharyngeal diverticulum in the production of pulmonary complications is not commonly recognized, Wilson[17] reports the case of a man 61 years old with a pharyngeal diverticulum which was causing difficulty in swallowing, "chest trouble," and "laryngitis" followed by a persistent cough. After excision of the diverticulum, which was located at the level of the upper border of the manubrium, the patient gained weight, and the cough, though still present, was not as persistent or productive as before operation.

Koch[18] presents the results from six months to 12 years after surgical treatment of pharyngoesophageal diverticula in 10 patients ranging in age from 48 to 82 years. In all cases the diverticulum was removed in one stage, under local anesthesia in nine and intubation narcosis in the other. Complications with pneumonia developed in three cases, resulting in death in one of these. Follow-up examination revealed absence of symptoms and no evidence of recurrence in all nine patients. Postoperative stricture requiring dilatation did not occur in any of the cases.

In the discussion of the surgical management of pharyngoesophageal diverticula, Vollenweider[19] describes the various stages of development of these pouches and discusses the symptomatology. Attention is directed to persistent cough, excessive secretion and a sensation of fullness in the throat. Dysphagia is largely determined by the size of the sac, its retention and the pressure exerted on the esophagus. Palliative treatment offers no hope, and Vollenweider warns of the danger of perforation from blind introduction of bougies into the esophagus.

Vollenweider advocates the two-stage operation for extirpation of the diverticulum; the second stage being performed two weeks after the first. During the last three years five cases were performed. These are briefly described.

The problem of accurate dissection of a pharyngoesophageal diverticulum may be difficult, and removal of the diverticulum and suture of the esophageal defect in the past has often resulted in a stricture at the operative site. Terry[20] recommends aspiration of the pouch followed by illumination of it with a specially designed light which is left in the pouch during the operation and passage of a large Magill tube into the esophagus preliminary to removal of the pouch. Intratracheal anesthetic is used. This technique has been employed successfully in five cases. Use of the esophagoscope for illuminating the pouch and fixing and dilating the upper part of the esophagus seems to us a much simpler and just as satisfactory procedure.

Negus[21] presents an excellent study of pharyngoesophageal diverticula, which should be read in its original form by all handling these cases. The evolution of the anatomy of the hypopharynx and upper end of the esophagus and its relationship to the larynx is completely presented. Negus gives the following factors as predisposing to the formation of a pouch: the low position of the larynx to mouth of esophagus in man; the arrangement of the oblique fibres of the inferior constrictor and the cricopharyngeus; the anterior attachment of the esophagus to the larynx; difficulties in coordination between the cricopharyngeus and the pharyngeal force pump and the absence of support at an unprotected spot. Since all other factors are present in everyone, the variable factor of incoordination of relaxation of the sphincter is given as the principal cause of pharyngoesophageal diverticula.

Early small pouches may respond to repeated dilatations of the cricopharyngeus and for technical reasons it is thought best not to operate on the small pouches. Negus prefers one-stage removal of the diverticulum but leaves the wound open to heal by secondary intention. No mention is made of hereditary factors in the etiology of diverticula.

Buckstein and Reich[32] present two cases of lateral pharyngeal diverticula. These sacs extend anterolaterally from the pyriform fossa and are thought to be due to unobliterated third and fourth bronchial pouches. They cause symptoms of dysphagia and regurgitation of undigested food. Diagnosis is made by spot films of the hypopharynx with barium swallow. No mention is made of indirect or direct laryngoscopic examination or of the treatment of this condition.

FOREIGN BODY.

Williams[33] reports the case of a man with chronic schizophrenia, aged 32 years, who swallowed a crucifix, 5 cm. by 3.2 cm., which lodged in the upper part of the esophagus and was succesfully removed under general anesthesia.

Wolfsohn[34] reports the case of a patient who ingested a wire foreign body which was removed from the upper esophagus within 24 hours. Several weeks later, mediastinitis developed, but this responded to antibiotic therapy. Two and one-half months later she complained of a clicking sound on swallowing and of regurgitation. Examination following ingestion of barium revealed a typical pharyngoesophageal diverticulum.

INSTRUMENTS.

Price[25] was prompted to construct an apparatus for aspiration of posterior pharyngeal secretions because of repeated failure to demonstrate eosinophilic cells in routine smears of nasal secretions obtained from sputum smears, smears of freshly swabbed secretions from the anterior nares and smears of nasal discharge or swabbings from the posterior nasopharynx. The apparatus consists of a shortened 20 ml. Fisher pipette of 0.6 cm. bore, a small rubber bulb, and a small length of flexible rubber tubing of 0.7 cm. diameter.

The smear is usually obtained about one-half hour after the patient has awakened in the morning. With two tongue depressors gently depressing the base of the tongue and so placed against the teeth as to prevent sudden closure of the mouth, the aspirator (with hand bulb compressed) is quickly slipped into the throat. Mucus is suctioned from the posterior pharyngeal wall into the pipette barrel, after which the apparatus is promptly withdrawn. Thin smears of the secretions thus obtained are immediately prepared on glass slides and when dry are examined microscopically.

IRRADIATION.

From 1939-1940, 1,365 white school children were examined at the Johns Hopkins University School of Medicine Otological Research Laboratory. A two-year follow-up was reported in 1942. As many of the children as possible were re-examined during 1946-1947. A comparison of the records of these two examinations is presented in a report by Guild.[26] The average interval between examinations was six and one-half years. All children had careful ear, nose and throat examinations including nasopharyngoscopy and audiometric studies. Some were treated for deafness by nasopharyngeal irradiation and others by surgical procedures. The conditions were the same at re-examination as at the original examination.

The largest group, 259 children, had normal hearing at the first examination and received no treatment. They showed a slight gain in hearing in the low frequencies and a slight loss in the high frequencies. A large number of this group had abnormal tubal orifices at both examinations.

The ears of 95 children who had impairment for high tones only were treated by nasopharyngeal irradiation. They showed the same gain in low frequencies as the untreated group and in addition a slightly greater loss for the high frequencies. About half of the 24 children with moderate hearing loss for all tones treated by irradiation showed moderate improvement in hearing for all tones.

We do not consider the evidence in this study to be conclusive with regard to the treatment of moderate deafness for all tones by nasopharyngeal irradiation. Perhaps the most useful information gained in this study is the normal change in hearing acuity over a six and one-half-year period in children; it would seem that this study would tend to decrease the indiscriminate use of nasopharyngeal irradiation in children.

Smith and Scharfe[27] present a brief review of the literature on irradiation treatment of deafness. They applied 50 mg. radium sulphate enclosed in a 0.3 mm. monel metal chamber to each side of the nasopharynx for eight and one-half minutes at two to three-week intervals for three treatments. Treatments were given to 50 patients; 28 had Eustachian tube obstruction, seven had mixed deafness and 12 had ears with chronic suppurative otitis media. From a study of their cases they were able to make several generalizations. Irradiation is effective in removing excessive nasopharyngeal lymphoid tissue but should be preceded by surgical removal of an adenoid mass if present. Other causes of Eustachian tube obstruction should be sought before irradiation is employed as it is not without danger. Improvement in hearing should not be expected in patients with nerve deafness or mixed deafness or in those in whom irreversible fibrous changes have occurred in the middle ear.

This article simply confirms the observations of others in the tremendous amount of literature published on this subject in the past few years.

A brief historical review of the use of irradiation in the pharynx and nasopharynx is presented by Lederer,[28] who believes that there is no danger in the use of nasopharyngeal irradiation if standard technique is followed. Best results are obtained in children with conduction deafness due to lymphoid hyperplasia around the Eustachian tube orifices. The importance of careful history taking regarding previous irradiation is stressed.

In the carefully controlled series of treatments with the 50 mg. monel metal nasopharyngeal radium applicator reported by Rubin and associates,[29] the amount of irradiation received by the physician was measured. Average exposure to the fingertips was 50 mr per treatment. Exposure to the remainder of the finger and the body was considerably less. Rubin and associates recommend 600 mr per week, the maximum exposure to the hands and fingers. On this basis, a maximum of 12 treatments per week should be the limit for any one physician.

An excellent set of instructions for personnel handling the radium applicator is given. It would seem well worthwhile to post these instructions in any office where this form of therapy is employed.

A study based on the use of irradiation of the nasopharynx with the standard 50 mg. radium monel applicator in 468 cases is presented by Bilchick and Kolar.[30] The calculated dosage is roughly estimated to be 2,860 r. at 1 mm. in 12½ minutes and is composed of 75 per cent beta and 25 per cent gamma rays. There is a tendency for patients to notice a dry, irritated throat 24 to 48 hours after operation and for pre-existing otitis media to flare up on the eleventh to thirteenth day, indicating the height of the erythema. The action of the irradiation is primarily on the lymphoid tissue, but changes in the epithelium of the nasopharynx also occur and here lies the potential danger of this therapy; however, no case of malignant change or other complication has been reported from the use of the radium applicator. Irradiation in this series was done on patients with conduction deafness, nerve deafness, postnasal discharge and several other conditions. Good results were obtained in cases of conduction deafness, particularly in children. Little if any benefit was obtained in its use for other conditions. Care in handling of the applicator is emphasized. It is advised that large amounts of lymphoid tissue can be removed surgically before irradiation.

Extensive experiments by Braestrup[31] were conducted to determine accurately the dosage of radiation delivered by a 50 mg. radium monel applicator. It was possible to measure both gamma and beta radiation. This was important as the monel applicator emits both; however, beta rays predominate only at the surface. These experiments indicate clearly that dosages commonly used in nasopharyngeal irradiation exceed those applied in any other type of radiotherapy for nonmalignant conditions; however, the high intensity is limited to a small volume of tissue close to the applicator. This explains why so few cases of radiation damage have been mentioned in the literature. This is a highly technical article and should be studied by all interested in dosage penetration of gamma and beta rays.

Lampe[32] warns that use of irradiation to the nasopharynx can conceivably be productive of harmful results in years to come, although following prescribed dosage there should be no danger attending its use. Radiation therapists employing radium have learned, sometimes by bitter experience, that radiation damage may become manifest years after a course of treatment which resulted in no apparent immediate harm. With nasopharyngeal beta irradiation, the potential biologic dangers must be considered both in relation to the physician applying the radium and in regard to the patient. Lampe warns that there is danger in repeating the applications of radium to the nasopharynx which greatly increases the potentiality of inducing severe vascular and connective tissue to the point where the tissue may become vulnerable to the insults of trauma and infection.

In a most interesting article, Day[13] does not deny that good results have followed use of irradiation; however, he does not believe that lymphoid tissue in the vicinity of the tubal orifice is responsible for tubal closure and deafness in the great majority of cases and he believes that irradiation of such tissue will seldom relieve deafness or cause the tubes to open. In spite of glowing reports of irradiation, Day has been greatly disappointed by his experience with this form of therapy. Two-thirds of all patients under his observation had a nasal allergy which had to be controlled before the aural symptoms were entirely relieved. Irradiation gave temporary relief to some of the allergic patients; but the tubes again became closed, and the deafness returned in six to nine months. The second most common cause of tubal obstruction in children, according to Day, is the presence of masses of adenoids or adhesions. It is surprising that only 10 per cent of cases of conductive deafness with tubal closure were corrected by removal of tonsils and adenoids. Day discusses prevention and prophylactic measures which should begin with the baby in its cradle and he believes that many hearing defects are due to improper feeding of babies. Unless the infant's head is elevated at least 30 degrees from the horizontal, it cannot swallow without forcing fluids into the nasopharynx. Day is of the opinion that on occasions the application of radium to the nasopharynx may be productive of harm, particularly if repeated too often. The nasopharynx should be examined carefully before the applicators are inserted, and the use of these applicators should be confined to conductive types of deafness. He describes a suggested experiment to which each user should subject himself so as thoroughly to appreciate the potency of the therapeutic agent. Day's article is replete with interesting data and common sense and should be read by all interested in this phase of therapy.

Bordley[34] emphasizes the fact that infected lymphoid tissue in the nasopharynx has long been recognized as one of the principal factors to recurrent acute and chronic infections of the upper air passages and ears. For years numerous surgical and medical procedures have been used unsuccessfully to clear up such infections. In 1924, Crowe and Burnam used irradiation successfully in a series of patients suffering from auditory obstruction caused by hypertrophied lymphoid issue in the region of the Eustachian orifices. This method of therapy has been accepted universally as having merit in selected cases. It is effective in treating conductive type hearing impairment resulting from chronic or intermittent auditory tube obstruction. The greatest care, however, must be exercised in the selection of patients for irradiation and Bordley discusses the various factors which influence this therapy. In analyzing the results of irradiation of the nasopharynx in these patients suffering from a conductive type hearing loss, he is convinced that improvement of hearing impairment is more successful in children than in adults. Fifty per cent of carefully selected children in Bordley's experience have shown significant improvement in hearing following irradiation. The effectiveness of this type of therapy in simple aerotitis media, as reported by Haines, showed that 90 per cent of submarine trainees suffering from aerotitis resulting from their escape tank test were able to resume training after irradiation. Great reduction in the number and severity of upper respiratory infections has been noted following irradiation. Bordley states that if the technique and dosage prescribed by Burnam and Crowe is followed accurately, no bad results should be obtained from the use of the radium applicator in the nasopharynx.

In an editorial on the nasopharyngeal applicator, Jacox[11] states that clarification of some problems associated with this applicator may be needed because of the recent observations of Braestrup on the ionization measurements of the tissue dose in equivalent Roentgens. The dosage employed thus far has been more or less empirical. Studies have been published which indicate that it would be well to monitor every physician's technique to determine the number of treatments he may administer without exceeding the permissible value. Jacox states that if the standard applicator is used with the recommended precautions, there is little likelihood of any serious damage. He points out that since it has been determined clinically that these exposures are safe and are necessary to obtain the desired results, radiologists should apply this knowledge of accurate ionization measurements in using the applicator in the future.

SURGERY.

Klopp and Delaney[36] point out that lesions of the hypopharynx, epiglottis, postcricoid region and base of the tongue are readily accessible through an anterior pharyngotomy. This procedure is simple, provides good exposure and does not interrupt any major vessel or nerve. A collar incision is made over the hyoid bone and the skin and platysma are undermined. The central portion of the hyoid bone is freed and a large segment removed. Incision of the pharyngeal mucosa then reveals a large area of the pharynx and hypopharynx and the base of the tongue for inspection and surgical procedure. The wound is carefully closed in layers without drainage. A perliminary low tracheotomy may be desirable. Postoperative healing is usually uncomplicated. Several illustrative cases are cited.

Farina[37] states that pharyngoneoplasties are indicated in patients in whom there is wide destruction of the pharynx. Pharyngectomies are generally performed on patients with cancer of the pharynx or advanced cancer of the larynx with invasion of the pharynx or perilaryngeal tissues requiring wide resection. Obviously, such extensive resection of tissue in the region of the neck would cause great discomfort because of constant discharge of salivary secretions which produce considerable local irritation of the skin. In addition to this, the patients experience much difficulty in swallowing and must be fed by a tube as long as the stoma is open.

The technique of the repair is well described and illustrated. Great stress is placed on the importance of nonsuperposition of suture layers to insure successful healing. Farina points out that the question of salivation and growth of hair in the interior of the pharynx no longer offers any serious obstacles for such repairs. The use of atropine is discouraged. Satisfactory results have been obtained by Farina. The patient is able to obtain nourishment in the normal manner and his psychic state is greatly improved.

This article is all too short to cover such an important subject. An illustrative case report is included and it is hoped that more cases will be reported.

BENIGN TUMORS.

Schall[38] correctly calls attention to the fact that the nasopharynx is the most neglected area in laryngology. It is an area difficult to examine in many adults and in children it is best examined under general anesthesia. Lesions occurring in the nasopharynx present symptoms varying in size, location and extension of the tumor. Schall discusses all symptoms under five headings: nasal obstruction, tubal obstruction, hemorrhage, invasion of cranial nerves and metastasis. Attention is directed to benign nasopharyngeal fibromas which at times present serious problems in adolescents. These fibromas are frequently found in children five or six years of age and rarely after 20 years of age. The origin of nasopharyngeal fibromas is in the prevertebral fascia at the base of the sphenoid. Such tumors may completely fill the nasopharynx. In the treatment of the highly vascular tumors, Schall states that Herman and Figi use electrocoagulation, but he much prefers irradiation, either external or by implantation of radium needles into the tumor. Such articles as this help to make the laryngologist more conscious of the pathologic alterations which may develop in the nasopharynx.

Diehl[39] presents a case of nasopharyngeal fibroma in a woman aged 52 years. It was successfully removed surgically with little bleeding and was believed to be a fibrosed angioma or polyp.

.Neurinomas are benign tumors arising from the neurilemmal sheath of peripheral nerves. They are firm and encapsulated and histologically show a palisading of spindle-shaped nuclei. They are rare in the pharynx, only about 15 cases having been reported. Kemmom[40] presents a case in which a large neurinoma occurred in the lateral wall of the pharynx which was removed through a pharyngeal approach. The involved nerve often degenerates following removal of the tumor.

Hunter and Biggart[41] report the case of a large nasopharyngeal fibroma, in a boy 17 years old, which produced typical symptoms. Deep Roentgen therapy, tried first because of the difficulties of surgical treatment, produced no change in the tumor. Complete recovery followed surgical removal.

Figi and Davis[42] present an analysis of 51 cases of nasopharyngeal fibromas, also known as juvenile nasopharyngeal fibromas, seen at the Mayo Clinic from 1940 through 1949. This is the largest series reported in the literature.

These tumors always appear during adolescence, and although they may show regression when the patient reaches sexual maturity, Figi and Davis have never observed complete spontaneous disappearance. These tumors are benign histologically but may be highly destructive by local invasiveness. They occur predominantly in the male. All patients in the series reported were males. The average age of their patients when first seen was 15.1 years and most of them had had previous therapy. The clinical findings are consistent. Nasopharyngeal examination reveals a red, firm, well rounded mass occupying the nasopharynx. Ulceration is unusual. The tumor may also be seen by anterior rhinoscopy. Figi and Davis believe that the diagnosis can be made by the gross appearance of the mass and biopsy is contraindicated because of possible hemorrhage. .

Treatment consists of electrocoagulation and implantation of radon seeds in most cases. One patient received electrocoagulation alone and seven, irradiation alone. Anesthesia is obtained by the intravenous administration of sodium pentothal and oxygen intratracheally. The tumor is approached through the nose and transorally through the nasopharynx and more recently transantrally through a Caldwell-Luc approach, in the latter instance the nasoantral wall is also removed Secondary operations are often necessary and are done after an interval of six to eight weeks

Of 42 cases with adequate follow-up, 72 per cent have been controlled There were no immediate operative deaths in this series. One patient died of meningitis three months, and one of hemorrhage four months postoperatively

This is an excellent and authoritative discussion of nasopharyngeal fibromas, and it would seem from the results obtained that the method of treatment described here is the procedure of choice

Switzer and co-authors[43] describe a case of extramedullary plasmocytoma with several lesions in the pharynx, in addition to the skin and lymph node involvement, which failed to respond to all known types of therapy These tumors are rare, their site of predilection is the upper respiratory tract. The clinically noncancerous stage may persist over 10 years, but it is ultimately fatal once the skeleton or lymph nodes become involved Irradiation may cure isolated lesions.

Scevola[44] reports two cases of plasmocytoma of the rhinopharynx The first case, a man aged 47 years with a fungating neoplasm on the lateral wall of the rhinopharynx, died two and one-half years after radiologic treatment with a large ulceration and glandular metastases The other case, a man 38 years old, had a fungating neoplasm on the arch of the rhinopharynx, which was treated radiologically after incomplete surgical removal, he was in good health three years following treatment

MALIGNANT TUMORS

Malignancies of the nasopharynx rank third in incidence among neoplasms in Singapore. Severe pain in the frontal and parietal regions in the later stages is believed to be due to extradural extension of the growth This pain often is not relieved by any drugs In four cases described by Mekie and Ransome[45] bilateral leucotomy was done for relief of the pain.

One patient died several hours postoperatively. The remaining three obtained remarkable relief of pain and did not show mental deterioration.

In an excellent article, Villoria[16] thoroughly discusses the subject of nasopharyngeal malignancies. According to him, prior to 1900, nasopharyngeal malignancies were considered extremely rare; since then, however, many cases have been reported, and it is generally thought to be more common than reported cases indicate. Regaud, of France, was the first to recognize nasopharyngeal tumors as lymphoepitheliomas. About this same time Schmincke described a similar type of tumor which he said was extremely radiosensitive. It was determined that lymphoepitheliomas and lymphosarcomas constituted the most frequent types found in the nasopharynx. Villoria describes a case of lymphosarcoma treated radiologically. Many patients with nasopharyngeal malignancies are probably seen by capable otolaryngologists who fail to make a correct diagnosis. The signs and symptoms of nasopharyngeal tumors are well described. These tumors have a tendency to infiltrate the orbit, base of the skull and cranial vault. The only two posterior methods by which a representative piece of tissue may be obtained for microscopic examination are either through the nose or by elevating the soft palate. The generally accepted therapy for nasopharyngeal cancer is radiation, as most of these tumors are radiosensitive. Villoria reports a case of plasmocytoma of the nasopharynx and practically reviews the literature on this tumor.

Owen[17] discusses at length the problem of hypopharyngeal carcinoma. The importance of early diagnosis is emphasized, including the significance of a collection of mucus in the pyriform fossa on mirror examination and esophagoscopy in cases of dysphagia. He believes that careful follow-up and repeated esophagoscopies are indicated in women with dysphagia and anemia, which he calls "Patterson-Brown Kelly syndrome." Of 34 such patients followed five years or more, postcricoid carcinoma developed in five. Owen prefers surgical treatment to Roentgen therapy. He differentiates the soft ulcerative type of postcricoid carcinoma which metastasizes early from the scirrhous type. He advises pharyngolaryngectomy with primary closure of the pharyngostomy with a skin graft for the ulcerative type, but believes that a lateral pharyngotomy or transthyroid pharyngotomy may be sufficient in the scirrhous type.

This article presents a good discussion of the problem of postcricoid carcinoma but adds nothing new to the treatment of this most distressing condition.

According to Eberhard and Leaming,[18] the ideal goal of radiotherapy for nasopharyngeal malignancies is a minimum of 4,000 to 4,500 r. to all parts of the tumor in four weeks. This is best accomplished by the use of two lateral fields and two antral fields, and in addition cervical fields when cervical metastases are present. All types of tumor should receive the same dosage when possible. Lymphosarcomas, which comprise 40 per cent, and lymphoepitheliomas, which comprise 10 per cent of malignant tumors of the nasopharynx, are more radiosensitive. In patients without bony involvement, about 40 per cent may survive five years; with bony involvement, only about 10 per cent will survive this period. An overall salvage rate of about 20 per cent should be expected from all patients treated by irradiation.

Reaction to treatment can be severe and medical management is of utmost importance. Occasionally because of a severe cutaneous reaction it is necessary to use radium locally rather than Roentgen-ray therapy.

This article contains exact descriptions of technique used which will be of interest to radiotherapists.

Quite a few cases of pituitary chromophobe adenomas have been reported in the nasopharynx, but Kay and co-authors[19] state that three cases described in this article are the first cases reported involving the nasal cavity. The diagnosis of all three cases was made by radiologic evidence of enlargement of the sella. These tumors do not metastasize but may be locally malignant and invasive. In each case nasal obstruction and rhinorrhea occurred. Rhinoscopic examination revealed reddish polyps in the postethmoid region in two cases and in the middle meatus of the third. The growths were partially removed intranasally in each case; the pathologic diagnosis was pituitary chrombophobe adenoma in two cases and it was questionable in the third. All three patients received Roentgen therapy and are living 14 years, five years, and 18 months after treatment.

Cunningham and Hu[50] briefly review the literature on nasopharyngeal cancer in the Chinese to direct attention to the prevalence of this disease in the Chinese between the ages of 30 and 40 years. An analysis of 37 cases which were encountered between 1944 and 1947 in the teaching hospitals of the West Chinese Union University showed that males were affected three times as often as females. In their series the most obvious sign was painless enlargement of the cervical nodes of the spinal accessory chain in one or both posterior triangles of the neck. Other clinical manifestations which indicate a possible malignant lesion in the nasopharynx are nasal bleeding, tinnitus and deafness, numbness over the maxillary region, paralysis of the VIth or IIIrd nerves, unilateral headache and trismus. Cunningham and Hu urge a nasopharyngeal examination for every patient with symptoms relating to the eye, ear, nose and neck.

Kramer[51] analyzed 54 cases of malignancy of the nasopharynx treated at Middlesex Hospital from 1935-1949. These tumors account for 0.7 per cent of the deaths from cancer in England.

Symptoms of these patients may be divided into three groups: symptoms due to presence of the tumor locally, those due to intracranial extension through the foramina of the skull, and those due to metastatic lesions, principally cervical nodes. Seventy-six per cent of the 54 patients in his series had cervical metastasis and 25 per cent had involvement of the cranial nerve when first seen.

Kramer differentiates squamous cell carcinoma, anaplastic carcinoma, transitional cell carcinoma, and lymphoepithelioma as well as the nonepithelial malignancies. It is of interest to note that over 50 per cent of these cases with cranial nerve involvement were transitional cell carcinomas; however, there is still much discussion among pathologists about the differentiation of the various types of malignancies occurring in this region.

The five-year survival rate in Kramer's series was 25 per cent. All patients were treated by Roentgen-ray therapy. Transitional cell carcinoma has the worst prognosis. Most of the patients surviving two years will be alive without recurrence at the end of five years.

In a clinicopathological conference[11] the clinical symptoms indicated the presence of a tumor of the nasopharynx invading the maxilla and the orbit. Biopsy from this tumor, which could be seen protruding from the nasopharynx, was reported as acute and chronic inflammation A medical man expressed surprise that he was asked to conduct the differential diagnosis instead of an otolaryngologist. A small ulcerative lesion on the scrotum proved to be a carcinoma and the Roentgenograms showed a questionable shadow in the mediastinum The white blood count was 56,000 per 100 cc. The disease was rapidly fatal, and autopsy revealed squamous cell carcinoma of the nasopharynx, maxilla and orbit A carcinomatous mediastinal tumor was found to have perforated into the wall of the superior vena cava, formed a thrombus and extended down into the auricle The tumor invaded the superior segment of the left lung and the lumen of the bronchus showed involvement. The question naturally arose as to which was the primary tumor. The pathologist expressed the opinion that this was a primary tumor of bronchogenic origin with metastasis to the nasopharynx, sinuses and cervical region

Such cases as this are of extreme interest to the otolaryngologist because they vividly demonstrate that at no time can we afford to confine our study only to the domains of otolaryngology. The symptoms described are typical of tumors of the nasopharynx and an alert otolaryngologist should have discovered the lesion long before it reached such alarming proportions This case further demonstrates that a negative biopsy report, in patients with clinical evidence of malignancy, such as cervical metastasis, is of no value and repeated biopsies should be done until representative tissue is obtained. Discussion of such cases is not only instructive but makes the otolaryngologist more conscious of disease of the nasopharynx.

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CAPILLARY AREAS OF THE COCHLEA IN THE GUINEA PIG.

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Interest in the vascular system of the inner ear was stimulated about the middle of the nineteenth century by the publication of the first histological descriptions of the end-organs within the membranous labyrinth. Investigators were impressed with the importance of understanding the circulation in such a highly differentiated structure and numerous attempts to solve the intricacies of the vascular configuration followed.

Most of the early investigators (Huschke,[1] Retzius,[2] Voltolini,[3] Boettcher[4]) noted the presence of blood vessels related to the inner ear structures which they studied and described their course and position, but Schwalbe[5] (1887) was the first to coordinate the circulation of the entire cochlea. The coiled arterioles of the modiolus particularly interested him, and he gave little attention to the capillaries. He was followed, in 1892, by Eichler[6] who introduced the technique of the intravascular injection of a dye and preparation of a cleared celloidin cast of the ear. While his studies on the human added little to Schwalbe's earlier work on the guinea pig, his methods were used in most subsequent investigations of the labyrinthine circulation.

Siebenmann's[7] illustrated monograph, published in 1894, became the classical study. He presented a clear picture of the general features of the circulatory pattern in man and

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emphasized the complete separation of the blood supply of the membranous labyrinth from that of its bony capsule. The capillaries in the spiral lamina and spiral ligament were shown as loosely organized networks.

Shambaugh,[8] in 1903, was the first to recognize an orderly arrangement of the vessels in the outer wall of the cochlea in the pig. He divided them into three distinct capillary areas. Further comparative studies by Asai,[9,10] in 1908, on the dog and the rat revealed nothing new except a similarity in the vascular pattern of these animals to that of man.

In 1923, Nabeya[11] published an extensive study of the labyrinthine circulation of the mammalian ear. He carefully outlined the more general vascular characteristics but added little to our knowledge of the capillary regions.

In recent years, only a few studies on specific capillary areas have been made. Belemer[12] (1936) reported that the vessels entering and leaving the stria vascularis were arterioles. In 1949, Agazzi,[13] investigating the vascular hydrodynamics of the spiral ligament in the guinea pig, divided the capillaries in this region into two separate systems.

Each one of these investigators has contributed something toward a better understanding of the circulatory pattern, but the study remains incomplete. Interest has been primarily centered about the larger features. The capillaries, in most cases, have been indistinctly shown and inadequately described. Little attempt has been made to locate them precisely in relation to various parts of the inner ear.

The necessity for further studies on certain vascular areas in the ear has been emphasized in the past few years. Recent reports have shown that there is still no clear concept of the capillary arrangement in the spiral ligament. The question of the origin and absorption of endolymph and perilymph is one that would profit from a more detailed analysis of the distribution of blood vessels. Ménière's disease, tinnitus, and some hearing disorders have been attributed to vasomotor disturbances in the cochlear and vestibular labyrinth. It is important that the details of capillary ramifications and relations be as clearly outlined as have other features of the vascular pattern. It is possible that a study of the capillary areas of the cochlea may add something to our knowledge of normal or pathological circulatory conditions.

MATERIALS AND METHODS.

Seventy-five guinea pigs weighing from 250 to 450 gm. were used. They were healthy animals of dark color with good pinna reflexes.

The vascular pattern was studied by means of the intravascular precipitation of Prussian blue or lead chromate. It was found that this method resulted in more complete capillary injections than perfusion of a dye or particulate material.

The animals were given 0.6 ml. veterinary Nembutal (Pentobarbitol Sodium, Abbott), the chest cavity was opened and a cannula inserted in the aorta. The blood was first washed out with 0.85 per cent sodium chloride solution or Locke's solution at approximately normal blood pressure.

An intravascular precipitation of Prussian blue was induced in 17 guinea pigs. This was accomplished by perfusing first with 75 to 100 ml. of 2 per cent of equal parts aqueous potassium ferrocyanide and iron ammonium citrate. A small amount (25 to 50 ml.) of distilled water was then run in to avoid precipitation in the tubing, followed by 50 to 75 ml. of 3 per cent hydrochloric acid in 10 per cent formalin solution. The temporal bones were removed from the animal, the cochleae were exposed and the specimens allowed to remain in the acidified formalin overnight. Some of these ears were completely decalcified, embedded in paraffin and sectioned. Most of the ears, after washing well, were dissected under the dissection microscope at magnifications of 18× to 54×. Pieces of the inner ear were removed as desired. They were counterstained lightly with 0.2 per cent aqueous Safranin O solution, dehydrated, cleared and mounted in Damar for microscopic examination.

The method of Williams[14] was followed for an intravascular precipitation of lead chromate crystals in eight guinea pigs. This method was found to be most suitable for visualization of the vascular pattern and topography of the cochlea as a whole under the dissection microscope at magnifications of 18× to 100×.

The cytology of the vessel walls was studied by the examination of dissected pieces of tissue stained with 0.2 per cent aqueous Safranin O, alone or combined with Light Green, and sectioned material stained by hematoxylin and eosin.

Bodian's protargol method (Silver albumose, Winthrop) was used on dissected and sectioned material to demonstrate the presence of nerve fibres. Eleven animals were perfused with 10 per cent formalin solution and the ears fixed for two days. Half of the ears were decalcified with 2.6 per cent nitric acid in 12 per cent formalin solution; the others were stained in the undecalcified state. The cochleae were opened, and the cochlear duct and blood vessels in the modiolus were exposed before silver impregnation. After impregnation the blood vessels were dissected out from the modiolus, pieces of spiral ligament and spiral lamina removed, and the staining procedure completed.

Sections of paraffin embedded ears were stained by Weil's iron hematoxylin method to differentiate between the myelinated fibres of the cochlear nerve and the unmyelinated fibres of the blood vessels.

FINDINGS.

The general features of the vascular supply has been previously described;[7,11] therefore, they will be outlined only briefly before going on to the detailed description of the capillary areas.

The labyrinthine artery enters the internal auditory meatus, gives off the A. vestibuli anterior, then continues to the base of the modiolus as the A. cochleae communis. Here it divides into the A. vestibuli posterior and the A. cochleae propria.

The A. cochleae propria enters the modiolus with the cochlear nerve. It gives off numerous branches in its spiral ascent to the top coil and ends somewhere in the fourth turn by dividing into two branches, one of which supplies the spiral ligament of the apex.

In its course upward, the A. cochleae propria is embedded in the loose connective tissue between the rope-like core of nerve fibres and the bony wall. In this position it gives off large primary branches which in turn ramify into secondary branches, or smaller branches equivalent to the latter. The primary branches may coil or twist a few times in the loose connective tissue before dividing further. The first primary branch is notable in that it is a large branch, often supplying a part of the basal turn and the vestibular portion of the cochlear duct, anastomosing with branches from the posterior vestibular artery.

The secondary radiating branches coil tortuously in bone before arching out over the scala vestibuli. They strike outward to enter a wedge-shaped plate of bone located superior and medial to the scala vestibuli, and inferior to the posterior spiral vein. In the bone, they assume the general pattern of a spring coil directed toward the bony partition; however, the coils are not completely enclosed in bony channels. At the root of the bony partition the arterioles straighten out, arching over the scala vestibuli enclosed in a bony canal for a variable distance to enter the spiral ligament at its uppermost attachment.

The distance for which the radiating arteriole traverses the bone usually decreases as the apex of the cochlea is approached. In the first turn the radiating vessel is encased in bone across the top of the scala, descends along the lateral edge and at times does not leave the bone until below the upper limits of the spiral ligament. In the second and third turns, the vessel leaves the bone after it passes the lateral limits of the bony partition. In the fourth turn and the apex, it may not be enclosed in bone for the greater part of its course. Wherever the vessels leave the bone before entering the spiral ligament, they are separated from the perilymphatic space by only the thin layer of mesothelial cells which lines the scalae.

I — Capillaries of the Spiral Ligament.

Although it may divide into two branches in the bony partition, the arteriole does not ramify into terminal branches until it enters the spiral ligament. One or two small branches turn and course in a spiral direction; the remainder descend in the spiral ligament. These capillaries of the spiral ligament may be divided into four groups on a basis of their location and course. Group 1 consists of the branches that wind in a spiral direction in the upper spiral ligament. Group 2 is the capillary network in the stria vascularis. Group 3 is the vessel in the spiral prominence. Group 4 includes the remaining capillaries which descend in the depths of the spiral ligament (see Figs. 1 and 2).

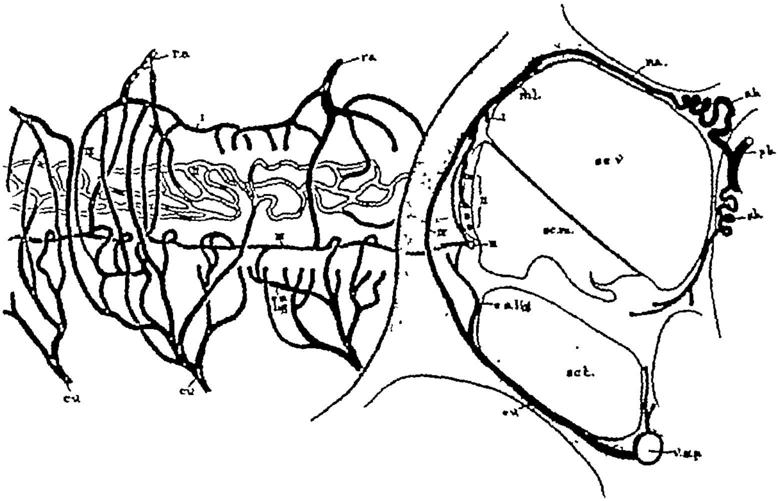

Fig. 1. Photograph of a schematic drawing showing typical capillary ramifications in the spiral ligament of the guinea pig. (c.s.lig.) crest of the spiral ligament; (c.v.) collecting venule; (m.l.) mesothelial layer; (p.b.) primary branch; (r.a.) radiating arteriole; (s.b.) secondary branch; (sc.m.) scala media; (sc.t.) scala tympani; (sc.v.) scala vestibuli; (V.sp.p.) V. spiralis posterior; (I) capillary of the upper spiral ligament; (II) network of the stria vascularis; (III) vessel of the spiral prominence; (IV) capillary descending in the depths of the spiral ligament.

Group 2: Each arteriole usually sends one of its branches to the capillary network of the stria vascularis. This is often a terminal vessel, but it occasionally divides, one part entering the stria vascularis, the other descending to the vessel of the spiral prominence or the lower spiral ligament. The branch descends in the spiral ligament behind the stria vascularis, usually entering the strial epithelium in its upper half to empty into the vascular plexus.

The capillaries of the stria vascularis pursue a serpentine course in the spiral direction in between the epithelial cells (see Fig. 3). They are extensively connected with each other so that they give the appearance of a network or wide one-layered honeycomb superimposed upon and disposed at right

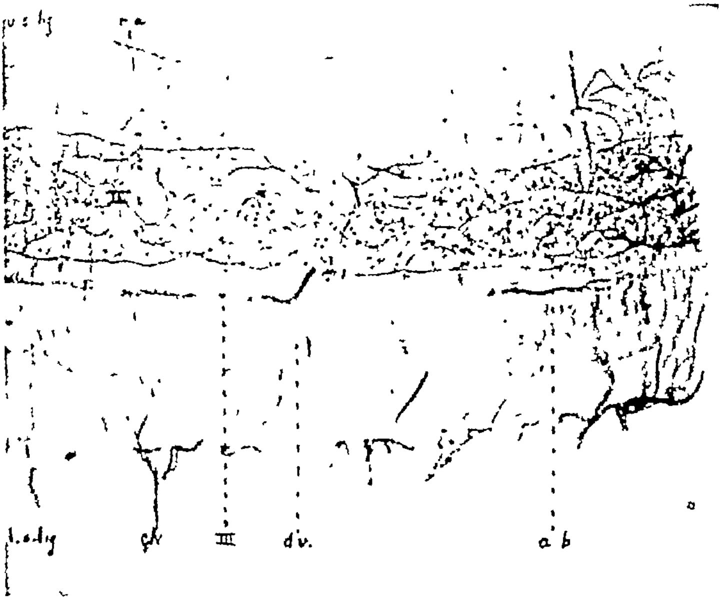

Fig 3 Photomicrograph of dissected specimen of spiral ligament from guinea pig No 21 showing network of the stria vascularis. Intravascular precipitation of Prussian blue, Safranin O stain; magnification 100×. (a.b) arteriolar branch entering the stria vascularis; (c v.) collecting venule; (d v.) venule draining the strial network; (l s.lig.) lower spiral ligament; (r.a.) radiating arteriole, (u s lig) upper spiral ligament; (II) network of the stria vascularis, (III) vessel of the spiral prominence

angles to the deeper vessels of the spiral ligament. The superior and inferior boundaries are comparatively straight and parallel.

The strial rete is drained by a venule of large caliber. It is formed by the junction of three or four of the strial capillaries and turns backward to leave the stria vascularis in its lower half. It descends peripherally to most of the other descending vessels and may receive a branch or two from them before entering into the collecting venous system at the lower edge of the spiral ligament.

In all of the animals observed, no other connection was noted between the strial capillaries and other capillaries in the spiral ligament except through the arterial branch entering and the venule leaving the network.

Group 3: Another of the main branches usually descends behind the stria vascularis and curves inward to enter the spiral prominence, where it empties into a spirally coursing vessel. Each radiating arteriole usually supplies one branch to the vessel of the spiral prominence, although there may be more than one, or none. The small capillaries of the first group seldom end here.

The vessel in the spiral prominence is found near the epithelial layer, sometimes with only a single layer of connective tissue cells interposed. It courses parallel to the network of the stria vascularis, just below its inferior border, but in no way connected with it. It is not continuous, rarely continuing for more than 1 or 2 mm. without a break. At times it is double, a duplicate vessel running for a short distance up under the edge of the stria vascularis.

This capillary is drained by many vessels, always two or three times the number of those entering it. They must bend upward and backward to leave the spiral prominence because of interference below from the outer spiral sulcus. They then descend toward the collecting venules of the lower spiral ligament.

Group 4: The remaining terminal branches descend in the spiral ligament, some curving out toward the periosteum. At times they divide in the depths of the tissue. This is found most often in the basal turn, where the tissue is thicker. Ordinarily there are no anastomoses between the capillaries descending in the spiral ligament until they reach the area below the basilar membrane.

Some of the capillaries descending close to the stria vascularis bend toward the scala tympani at a point from 30 to 50 microns below the level of the basilar membrane. They turn and continue in a spiral direction for 50 to 400 microns, sometimes receiving other of the descending vessels. In the basal turn this part of the scala tympani is covered by a thin plate of bone. Elsewhere, the vessels lie close to the mesothelial lining of the scala, with only a few descending basilar membrane fibres intervening. When the capillaries again turn downward, they continues to the collecting venule, lying below the mesothelial layer.

The other descending capillaries, following the curvature of the bone, turn slightly inward to meet the vessels just described, those from the strial rete and the spiral prominence. They are all united in the venules of the lower spiral ligament. At the inferior boundary of the scala tympani, they have been brought together into two or three trunks. These then cross the floor of the scala tympani, slanting in a basal direction, uniting into one large vein. Corresponding to the situation of the radiating arteriole, the venule does not enter the bone of the floor of the scala in the upper turns until it has almost reached the posterior spiral vein.

The V. spiralis posterior receives all the venous blood from the cochlea. It begins in the apex by receiving venules from the spiral lamina and spiral ligament, and makes a spiral descent at the inferior medial border of the scala tympani. As it descends it receives veins radiating inward under the scala tympani from the spiral ligament, those descending from the spiral lamina and others from the spiral ganglion and modiolus. At the proximal end of the basal coil, it turns and enters the V. canaliculi cochleae at the mouth of the cochlear aqueduct. The V. fenestrae cochleae and the V. vestibuli posterior, both of which may collect a few venules from the vestibular portion of the cochlear duct, receive drainage from the remaining part of the membranous labyrinth. They empty into the V. canaliculi cochlea which terminates in the jugular vein.

II — Capillaries of the Spiral Lamina (see Fig. 4).

The vessels supplying the spiral lamina originate either from the primary branches of the A. cochleae propria or the

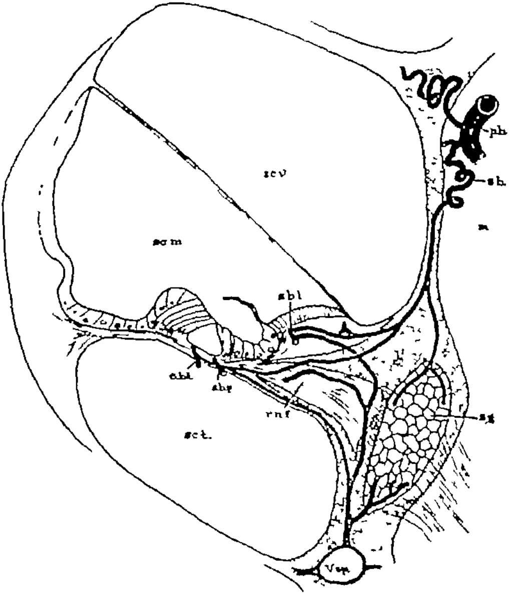

Fig. 4 Photograph of a schematic drawing showing typical capillary ramifications in the spiral lamina of the guinea pig. (m) modiolus; (p b) primary branch; (r n.f.) radial nerve fibres; (s.b.) secondary branch, (s b.l.) spiral border of the limbus; (s.b p.) spiral border below the inner pillar; (s.b.t.) spiral border below the tunnel; (s.g.) spiral ganglion; (sc.m.) scala media; (sc.t.) scala tympani; (sc.v.) scala vestibuli; (V sp p.) V spiralis posterior.

proximal end of the secondary coiled branches. They may coil a few time in the modiolus, then descend toward the spiral lamina. They turn outward in the upper plate of the bony spiral lamina to slightly beyond the medial limits of the limbus. If they have not previously branched, they divide here in various ways. One tributary may ascend into the limbus, while another continues outward in the bone. At times the vessel divides into two divergent branches coursing spirally at the root of the limbus, sending capillaries into the limbus and finally entering there itself.

The capillary bed of the spiral lamina is divided into two regions for descriptive purposes: an upper plexus in the limbus, a lower plexus below the basilar membrane. Each plexus may have separate arterial supply and venous drainage, although some capillaries are continuous between the two.

Vessels of the Limbus: The vessels enter the limbus by passing into the connective tissue base near its medial attachment. They ascend diagonally toward the lateral edge of the epithelial covering. The capillaries do not enter the epithelial layer, nor the fibrous area bordering the internal spiral sulcus but remain in the connective tissue base of the limbus. At the superior lateral edge of the connective tissue, inferior to the epithelial layer, the vessels turn and form an irregular edging of loops (see Fig. 5).

From the upper edge of the limbus, the vessels descend in the same manner through the connective tissue to re-enter the upper plate of the spiral lamina. In the bone, the capillaries either continue alone toward the vein or join the descending vessels from the lower plexus.

Vessels Below the Basilar Membrane: The arteriolar branch to the plexus situated below the basilar membrane ordinarily approaches in one of two ways: either by descending in the bone of the modiolus and continuing out in the bony spiral lamina after giving off a tributary to the limbus, or by descending in the central canal of the modiolus. The first manner of approach has been described. The second manner of approach is found in the first turn, part of the second and

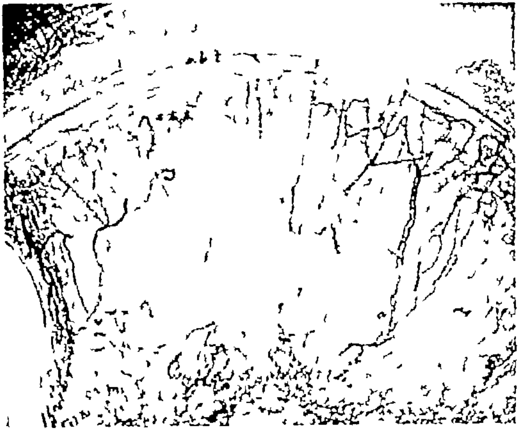

FIG 5 Photomicrograph of dissected specimen of spiral lamina from guinea pig No. 66 showing the capillaries of the limbus Intravascular precipitation of Prussian blue Safranin O stain magnification 65X (a) arteriole; (l) limbus (m) modiolus (s b l) spiral border of the limbus (s b t) spiral border below the tunnel (v) venule

infrequently elsewhere The arteriole twists as it descends in the modiolus, then pierces the superior medial corner of the bony wall of Rosenthal's canal Here or before, branches may be given off to the limbus and spiral ganglion

Whatever the manner of their approach to the upper plate of the spiral lamina, they extend outward either enclosed in the bone or placed directly on top of the radial fibres of the cochlear nerve Here the vessels are in intimate contact with the myelinated fibres In spreading out they sometimes dip under a few fibres They descend to the lower plate of the spiral lamina through the nerve, usually traversing the interspaces, but at times penetrating the bundles

Having reached a position inferior to the nerve fibres, the vessels continue out beyond the bone and below the fibres of the basilar membrane Below the inner pillar of the organ of arteriole descending to the spiral lamina. The vessels enter Rosenthal's canal from above and intertwine freely among the ganglion cells. There is little anastomosis with the capillaries leaving the basilar membrane and limbus. At times the vessels are collected together in the same postcapillary venules. Otherwise the venules from the spiral ganglion enter the posterior spiral vein by a separate collecting venule.

The arterial supply to the spiral ganglion appears to be entirely separate from that to the fibres in the central canal of the modiolus. In the proximal part of the first turn, the fibres leaving the spiral ganglion are enclosed in bone for a short space from spiral lamina to central canal. The vessels supplying these fibres sometimes make connections with the spiral ganglion vessels and even those leaving the basilar membrane; however, in many cross-sections observed, no vessels were ever noted crossing the bony plates between the ganglion cells and modiolus, or associated with the fibres leaving the spiral ganglion, other than the exception above noted.

The spiral nerve fibres in the central canal of the modiolus are well supplied by vessels coming directly from the artery or from the primary branches. They are highly anastomotic and uncoiled. The collecting venules enter the posterior spiral vein from the modiolus.

IV — Cytology of the Vessel Walls.

The thin vessel wall of the A. cochleae propria in the central canal of the modiolus contains two or three layers of longitudinal and transverse smooth muscle cells around the endothelial lining, plus a narrow adventitia. Its tributaries, decreasing in size, have a relatively less dense muscularis. As the vessels coil outward in the bone, the muscle cells are found to be more and more sparsely spread.

The arteriole radiating out over the scala vestibuli consists of an endothelial lining, with an occasional tangental or longitudinal—more rarely transverse—smooth muscle cell around the endothelial layer and a thin adventitia of two or three layers of connective tissue cells arranged parallel to the vessel. A small accumulation of transverse smooth muscle cells can sometimes be observed in the fork or around the arteriole as it divides in the upper spiral ligament. The perivascular cells are divided between branches so that each main branch is usually enclosed in one layer of connective tissue cells, longitudinally disposed. When the main branches again divide, the connective tissue coating is lost.

The capillaries are composed of endothelial cells and an infrequent smooth muscle cell. This is typical of all of the capillaries of the spiral ligament except those in the stria vascularis. As the collecting venules leave the spiral ligament, a connective tissue adventitia is once more added.

The capillaries of the strial network appear to be composed only of endothelial cells. In some preparations, cells were seen which seemed to be applied to the outside of the capillary wall, but whether they were smooth muscle cells or closely applied epithelial cells could not be determined with certainty.

The vein draining the strial rete, as before noted, is of large caliber. It was observed at times to have one, two or three smooth muscle cells arranged transversely or tangentally across its mouth. The venule obtains a connective tissue perivascular layer as it leaves the stria vascularis. This is not characteristic of other vessels at this level.

The vessels supplying the spiral lamina are similar in appearance to those of the spiral ligament. The arterioles approaching the spiral lamina gradually lose most of their smooth muscle coating. As they enter the limbus and Rosenthal's canal, they are reduced to capillary size. The capillaries extending throughout the spiral lamina are endothelial walled tubes with occasional smooth muscle cells applied about them, particularly at bifurcation points. The vessel below the tunnel appears to contain fewer contractile elements than the other capillaries; however, the oval nuclei of the cells of the basilar membrane are closely applied to this vessel, at times giving impression of being directed around the vessel wall.

The capillaries in the spiral ganglion and those supplying the nerve fibres in the modiolus are likewise endothelial walled tubes, with occasional smooth muscle cells.

V — Innervation of the Blood Vessels.

Many nerve fibres accompany the labyrinthine artery entering the internal auditory meatus. Some are found in bundles contained in a connective tissue syncytium. Numerous naked nerve fibres can also be seen, either in the vessel wall or closely applied to it as it enters the meatus. The fibres are unmyelinated and seem to have no connection with the cochlear nerve.

The fibre bundles become smaller as they follow the artery up into the modiolus, branches being sent out from the bundles to the artery and its primary and secondary tributaries. The nerve fibres were found on the coiled secondary branches of the cochlear artery, but no fibres were demonstrable with the arterioles radiating out in the bony partition, or descending in the bone of the modiolus to the spiral lamina; neither were any nerve filaments observed with the capillaries.

No definite specialized endings could be observed. As the fibres become closely applied to the vessel wall, they lose any sheath in which they were previously enveloped and disappear as naked fibres.

DISCUSSION.

The capillaries of the spiral lamina have received consideration previously by all investigators, and little can be added concerning the general pattern. Attention has been directed primarily to the spiral vessels which have been described in various places in the spiral lamina. Shambaugh[8] objected to the term "spiral vessel." He emphasized that these were borders of capillary areas formed by capillary loops anastomosing with each other. Nabeya[11] recognized a similar condition in the human, designating only the vessel below the tunnel as a spiral vessel. "Spiral border" seems to be a preferable term, as "spiral vessel" infers a vessel continuous from base to apex The spiral borders found in the limbus and below the basilar membrane are discontinuous and often present a looped appearance

The relation of the two lower spiral borders to the basilar membrane should be clarified Retzius stated that the spiral vessel below the tunnel was located in a layer of basilar membrane cells below the basilar membrane fibres Boettcher[1] described the vessel under the tunnel as being below the basilar membrane Siebenmann located it in the basilar membrane Asai[8] agreed with Siebenmann Nabeya[11] found two spiral vessels on the basilar membrane Observations made on the present series also revealed two spiral borders, one below the inner pillar cell, and one under the tunnel They were found to be beneath the basilar membrane fibres with one exception The vessel below the tunnel in the basal turn of some guinea pigs seemed to have a thin layer of basilar membrane fibres coursing beneath it so that the vessel was between the fibres This situation was observed only in the first turn

A description of all the capillary ramifications of the spiral ligament has not previously been given Shambaugh,[6] in 1903, first showed a definite arrangement for the capillaries of this region in the pig He recognized a capillary area over the scala vestibuli which he believed to be intraosseous, another in the stria vascularis, and a third in the deeper part of the spiral ligament. He described the strial capillaries as a continuous system of loops, separate from the other vessels in this area Nabeya[11] believed the capillaries of the stria vascularis to be superimposed upon those of the spiral ligament. Agazzi[13] divided the vessels of the spiral ligament into two systems one, the capillary rete of the stria vascularis, the other consisting of the short, direct vessels in the depths of the spiral ligament

The capillaries of the spiral ligament can actually be classified into four groups one in the upper spiral ligament, one in the stria vascularis, one in the spiral prominence and a fourth in the depths of the spiral ligament. A typical secondary radiating arteriole divides in the upper spiral ligament and sends one or two branches to the first group, one to the stria vascularis, one to the spiral prominence and several to the last group.

The small capillaries in the upper spiral ligament wind in a spiral direction and are not intraosseous as Shambaugh[8] found them to be in the pig. In the guinea pig they are situated just below the mesothelium lining the scala vestibuli. Some of them do traverse the bone as they ascend to the vein of the turn above.

The capillary network in the stria vascularis usually has one arteriolar branch entering it and a venule leaving it at approximately equivalent intervals. It has no other connection with the capillaries of the spiral ligament. This was indicated by Shambaugh[8] and Agazzi.[13] The system is composed of a closely meshed band of capillaries continuous from vestibule to apex.

The vessel of the spiral prominence has been given little attention. Shambaugh[8] included it in his illustrations as a part of the strial rete. Nabeya[11] noted the presence of a vessel in the spiral prominence but did not describe it, while Agazzi[13] did not include it in his model of the vessels in the spiral ligament. It was found in these studies that the vessel in the spiral prominence was a constant feature. While it is not continuous from base to apex, it usually can be found coursing straight through the prominence for 1 to 2 mm. without a break. Although it courses just below and parallel to the inferior border of the strial rete, it has no connection with the network of the stria vascularis.

The vessels descending in the spiral ligament were designated by Agazzi[13] as short, direct arteriovenular arcades. This applies to all except those that turn in the spiral direction for a short distance in the crest of the spiral ligament. Nabeya[11] described a spiral vessel in this area in the guinea pig. Asai[9] also noted a spiral vessel in the crest of the spiral ligament of the dog. Siebenmann[7] believed it to be an inconstant vessel with a spiral course. Shambaugh's[8] observations are in close agreement with those of this study. No spiral vessel was observed in this area for the guinea pig. Some of the capillaries descending in the depths of the spiral ligament curve toward the scala tympani. Reaching the surface of the crest of the spiral ligament, they turn to course in a spiral direction for 100 to 400 microns, sometimes receiving branches from other capillaries. Eventually, they descend to the venules.

Few observations have been made as to the structure of the vessel walls. One of the early investigators, Schwalbe,[4] described the smooth muscle coating of the coiled secondary branches in the modiolus. Little has been said about the vessels elsewhere. Belemer[12] reported that vessels entered and left the stria vascularis as arterioles. Agazzi[13] called the vessels in the depths of the spiral ligament arteriovenous arcades but specified that they were endothelial walled tubes unprovided with smooth muscle cells.

The capillaries in the spiral lamina and the spiral ligament with the exception of those in the stria vascularis, all revealed scattered smooth muscle cells. Only the capillaries of the strial network showed no definite contractile elements. These appear to be composed only of endothelial cells.

The only observation on the innervation of blood vessels in the inner ear was made by Lorente de No,[14] who found nerve fibres passing from the cochlear nerve to the blood vessels in the modiolus of the rat. He could not determine the extent of these fibres but believed they were limited to the vessels in the modiolus. Contrary to his findings in the rat, numerous nerve fibres were found accompanying the labyrinthine artery in the internal auditory meatus in the guinea pig. They seemed to have no connection with the cochlear nerve. The nerve fibres were apparent only in relation to the vessels of the modiolus.

SUMMARY.

These studies were initiated with the purpose of filling in the details of capillary ramifications and relationships within the cochlea. By perfusion and special staining methods, the vascular pattern, the cytology of the vessel walls and innervation of the vessels have been studied in the guinea pig.

The capillary pattern in the spiral ligament has been described and the vessels of the region divided into four groups on a basis of course and location: one in the upper spiral ligament, one in the stria vascularis, one in the spiral prominence and a fourth in the depths of the spiral ligament.

The capillaries in the spiral lamina have been studied with an emphasis placed on their relationships and connections.

The capillaries of the stria vascularis appear to be composed only of endothelial cells. Smooth muscle cells were observed in the vessel walls of all other capillaries in the cochlea.

Nerve fibres were found to accompany the labyrinthine artery in the internal auditory meatus and to continue with the A. cochleae propria and its branches in the modiolus. No nerve fibres could be demonstrated with the capillaries of the spiral ligament or spiral lamina.

My sincere gratitude is expressed to the members of the Departments of Anatomy and Otolaryngology of Washington University School of Medicine, and the Research Staff of Central Institute for the Deaf for their constant interest and guidance.

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ACROMEGALY.

With Special Reference to the Otolaryngological Aspect.*

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INTRODUCTION AND MATERIALS

A recent report of the occurrence of bilateral abductor paralysis with acromegaly and the admission of such a case to the infirmary stimuated the review of 48 cases of acromegaly that were studied at the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital since 1935. These cases were reviewed with special attention paid to the otolaryngological picture. Passing comment is made of the general signs and symptoms, laboratory findings and treatment. This paper does not attempt to make a complete medical study of the disease. The findings should be of interest to the internist, the endocrinologist and the surgeon as well as to the otolaryngologist. They will point out the necessity of a complete otolaryngological examination in all cases of acromegaly.

To summarize the literature — in 1907, Neufeld published a bibliography of the work in this field up to that date; from 1907-1928, the literature is reviewed and summarized by Freystadtl; after 1928, five papers as listed in the bibliography have been published.

Signs and Symptoms:

The patients uniformly complain of general lassitude and weakness. Although there is the general increase in the size

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of the bones and a progressive coarsening of the features, the patients do not seem to notice it until the condition is far advanced. They may first notice that there is an increase in the hat or glove size. In patients with false dentures, failure of the plates to fit properly may be the first complaint. Malocclusion and inability to chew properly because of the marked prognathism may occur in patients with full dentures

Otolaryngological:

Headaches occur in 70 per cent of the patients. The onset of the pain is in the occipital region with radiation to the frontal area At first they are mild and fleeting, but with progression of the disease they become intractable The pain is more severe in the morning and tends to diminish toward evening.

Face pain was present in 23 per cent of the patients. Only two of the cases showed causes other than the pituitary adenoma to account for the presence of the pain

The distribution of the pain is over the maxillary division of the Vth nerve and in three cases was so severe that the *nerve had to be sectioned for relief* Other cases were improved temporarily with alcohol injections of the nerve. Less often the face pain occurred over the ophthalmic distribution of the Vth nerve

Another type of face pain may occur this is the dull, gnawing pain that occurs over the jaw It is explained by the rapid growth of the mandible with the resultant pressure on the nerves

Laryngeal complaints were present in one-third of the patients. They varied from hoarseness and lowering of the voice to severe cough and dyspnea. There was one case of bilateral abductor paralysis in this series

Difficulty in swallowing was present in five patients Except for the generalized muscular weakness, no explanation could be found.

EXAMINATION.

There are many complete descriptions of the appearance of the far advanced changes in the acromegalic patient due to the effect of pituitary hormones which stimulate the growth of all tissues. There is a gross appearance of the face and typical acromegalic facies and habitus. The lower lip protrudes, the nose is large and broad, the expression is dull, and the supraorbital ridges are prominent. The teeth are short and often absent, due to the pressure of the bony growth. The tongue is characteristic. It is large, freely mobile, thick, and covered with coarse furrows. The mucous membrane of the oral cavity is hypertrophied and thick. The epiglottis is enlarged in all dimensions. The larynx, too, is enlarged, more in the lateral measurement, and the membranes are thick and redundant. Marie P. and Marinesco G. studied microscopical sections of these membranes in patients with acromegaly and reported that the changes found resemble those of pachydermia of the skin. The true cords are thick and white and move loosely with phonation.

Nasopharyngeal examination should be routine in these cases since the tumor may destroy the sphenoid and project into the nasopharynx. In the one case in this series where this was suspected the mass proved to be adenoid tissue.

It is in the examination of the sinuses that the otolaryngologist may be the first to discover the presence of a pituitary tumor. In over 85 per cent of the cases, the sinuses were abnormally large and well aerated. Transillumination may cause this to be suspected. Examination of the visual fields showed a bitemporal hemianopsia in 30 per cent of the cases.

Laboratory — X-ray changes are present in 90 per cent of the cases. They vary from showing merely enlargement of the sinuses and extensive prognathism to complete destruction of the clinoids.

With the adenomatous growth of the pituitary gland, there are accompanying adenomatous growths in many other organs. Many tests to determine the presence of these growths have been devised by the endocrinologists. A few of these studies are done periodically to determine the current status of the growth, to determine progression of the disease and to determine the effects of therapy. Some of the more routine studies are:

Serum phosphorus and phosphatase — Elevated.
17-Keto-steroids — Elevated.
B. M. R. — Elevated.
Blood sugar — Elevated.

Treatment:

Treatment of pituitary adenoma may be divided into three main groups — hormonal, X-ray and surgical.

Hormonal therapy was used in 15 per cent of the cases with some benefit. Recurrence and progression of symptoms was the rule and other treatment was necessary. ACTH was used in several cases, but the results thus far are inconclusive. Over 50 per cent of the cases were treated with X-ray. The average dose was 200 k.v. with a total dose of 1,000 r. through three portals, two lateral and one frontal. Two-thirds of the patients treated with X-ray were improved, but repeated courses of treatment were required. One patient received a total of 14,800 r.

The transphenoidal removal of the pituitary as done by Oscar Hirsch in selected cases offered the best chance of a complete remission of symptoms. Five cases done with this technique by Hirsch were improved. The neurosurgical approach, craniotomy, as done by the neurosurgical department of the M. G. H., gave good results in two cases.

Discussion:

Pituitary adenoma causes a large and varied group of clinical findings. The otolaryngological picture has been neglected because of the striking changes in the rest of the body. As pointed out by Grotting and Pemberton, if patients with acromegaly have a cord paralysis that is not found before operation it may endanger the successful outcome of the procedure. These authors reported five cases of bilateral vocal cord fixation (or paralysis) in acromegaly. Oscar Hirsch in a personal series of over 300 cases of acromegaly has not seen one case of bilateral abductor paralysis. The records of the infirmary do not mention any cases of cord paralysis except for the case reviewed. The cause of the bilateral vocal cord paralysis in this case may be explained by the presence of the mediastinal mass with pressure on the recurrent nerves. New and Childrey, in a review of 217 medical cases, found that malignant or benign enlargement of the thyroid may cause paralysis of the vocal cords. In their series there were 32 cases of cord paralysis from benign and 10 cases from malignant goiter. In two of the 42 cases, the paralysis was bilateral. Since the enlarged thyroids have been found to occur often in acromegaly, recurrent nerve pressure is probably the most logical explanation for the cord paralysis. It is definite that all patients with acromegaly should have a complete laryngological examination.

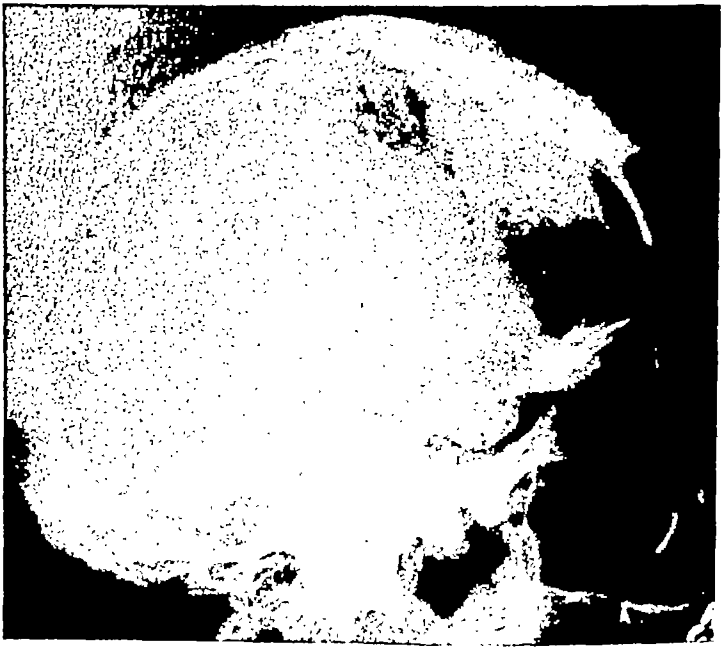

Fig. 1. Lateral view. X-ray showing typical skull findings in acromegaly: note abnormal enlargement of sinuses, thick cortex, ballooning of sella turcica, destruction of anterior and posterier clinoid proccesses.

CASE REPORT.*

A. J. 450115. The patient, a 73-year-old Russian and a former insurance agent, was admitted on March 19, 1951, with a chief complaint of respiratory difficulty for one year's duration with a marked increase in the severity of the symptoms for one week preceding admission. Over the past year, orthopnea, exertional dyspnea and ankle swelling had been present. Stridor, which was quite marked on admission, had been present for one week.

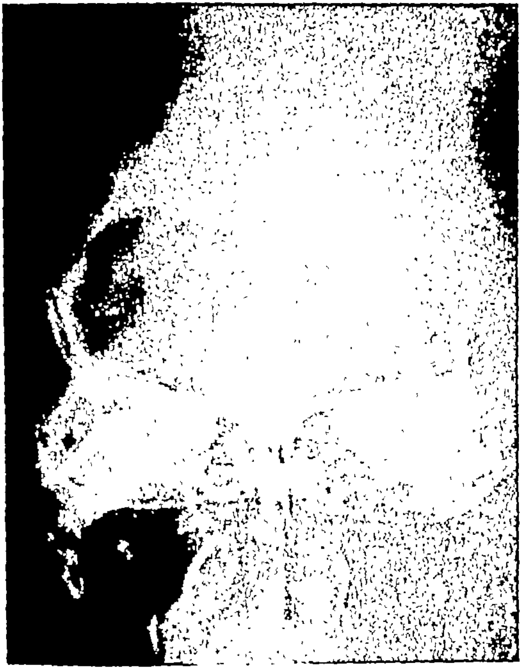

Fig 2. A-P view

Presented at the meeting of the New England Otolaryngological Society March 28, 1951. This case was included in a paper by Bauer and Aub bliography), in which particular attention was paid to the endocrinoical aspect.

In 1944, the patient had presented himself at the throat clinic complaining of nasal obstruction, and a diagnosis of bilateral polypoid sinusitis was made. A routine laryngeal examination at that time revealed a left-sided abductor vocal cord paralysis for which no cause could be found. In 1946, both cords were paralyzed, but the patient had few complaints of respiratory difficulty. Until the current admission the patient was followed in the throat clinic. The advanced age of the patient prevented sinus surgery.

Past History: In 1926, the patient began to notice changes in the appearance of his face. His attention was first called to these changes by a friend who remarked that he looked like an old man and that his features were becoming coarse. In 1934, the patient noticed that his lower jaw was becoming more prominent and that his lower teeth overlapped his upper teeth. At about this time his voice became deeper. There were no complaints of headache, visual disturbance or personality changes. He later developed glaucoma which obscured any picture of temporal hemianopsia which may have occurred. The general physical condition of the patient, except for the marked weakness which accompanied his acromegaly, was fair. There had been complaints of orthopnea and exertional dyspnea since 1935. In 1936, a BMR was plus 26. On treatment with Lugol's solution it fell to plus two.

Previous Operations and Treatment:

1930—Excision of multiple fibropapillomata from neck and occipital region.

1935—Sialolithiasis with removal of the left submaxillary gland.

1936—X-ray treatment to pituitary adenoma with remission.

1944—Nasal polypectomy and removal of inferior turbinate.

1947—Chronic glaucoma.

1949—Removal of senile cataract.

Physical Examination: The patient showed typical acromegalic facies. Marked emphysema and kyphosis were present. Mild congestive heart failure was present with scattered rales over the bases of both lungs and a two plus pitting edema of the extremities. The nose was markedly enlarged. The naris on the left was filled with polyps and pus. The tongue was large, red and mobile, the larynx was large and the mucous membrane loose, thick and lusterless. The vocal cords were paralyzed in adduction with less than 1 mm. of airway. The excessive secretion and pus which had collected on the surface of the cords made the airway even smaller. The false cords were hypertrophied and the true cords were thick and white.

Laboratory Reports: Blood and urine within normal limits.

March 22, 1951—Serum calcium, 9.9. Phosphorus, 4.6. Phosphatase, 3.2.

March 20, 1951—Electrocardiogram—Auricular flutter, left ventricular hypertrophy.

Bacteriology—Nasal culture—Coagulase positive hemolytic staphylococcus aureus, E. coli.

X-ray:

March 19, 1951—Fluid in right base. Mass in superior mediastinum.

March 19, 1951—No obstruction to swallowing.

March 20, 1951—Skull bone thick and heavy, frontals large. Sinuses—
Chronic purulent sinusitis, marked reaction around
frontals and antrum.

In 1936, there was a report of large bones of hands, clubbing of phalanges, coarse trabeculation of bones. Sella enlarged and floor depressed.

Hospital Course: On admission, March 19, 1951, the patient was in severe respiratory distress, and a tracheotomy was done under local anesthesia. The patient was digitalized, and the cardiac congestion that was present on admission was relieved. Local treatment, including a shrinking spray and local irrigations was instituted in an attempt to improve the chronic sinusitis; chemotherapy was also given. The foul discharge gradually diminished and the nasal polyps have disappeared. Two days after admission the patient had difficulty in voiding and was transferred to the genitourinary service for treatment of an enlarged prostate. The patient withstood prostatectomy well. He has been discharged from the hospital and is being followed in the out-patient clinic. The tracheotomy tube is still in place and the patient has no complaints. It was concluded that the substernal mass probably represented an enlarged thyroid as it was present in films taken in 1936.

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MULTIPLE PAPILLOMA OF THE LARYNX:

A PRELIMINARY REPORT OF FOUR CASES TREATED WITH TERRAMYCIN.

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Papilloma of the larynx is the commonest laryngeal tumor of childhood. Although they are benign, the mortality rate may exceed that of carcinoma of the larynx in adults.[1]

Microscopically, these tumors are identical to the papillomas that occur in adults; however, they differ in several important aspects and should be considered as a separate clinical entity.

The incidence of cases has been reported as between one per 1,000 to one per 6,000 clinic patients.[2] The symptoms may occur from birth onwards; more commonly they develop before the age of five years. The sex incidence is about equal, and the racial incidence compares with that of the general population.

The etiology of the disease is unknown. Heredity and chronic iritation have been blamed, but there is little support for these theories. It has been repeatedly observed that papillomas in children tend to disappear at puberty, which suggests some hormonal influence; however, while the lesions tend to disappear in response to the hormonal changes of puberty, it does not necessarily follow that they occurred because of some hormonal influence or imbalance in childhood. Certainly, there are usually no other signs or symptoms to suggest such a disorder.

In 1923, Ullman[3] demonstrated what was presumed to be a virus in the laryngeal papillomas of a child, by two successive

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transfers of the lesion to the skin of adult volunteers. He also made three attempts to transfer a solitary papilloma of an adult to healthy skin, but without success.[4]

In 1933, Shope,[5] using material obtained from the skin papilloma of wild rabbits, was able to demonstrate that an agent which passed Berkefeld filters of V. N. or W. porosity was capable of producing similar lesions in the skin of other rabbits and was further able to demonstrate a neutralizing antibody to this agent in the infected animals. Rous,[6] using the Shope virus, was able to produce papillomas in the muscle and the viscera by direct implantation, and in the lungs by intravenous inoculation.

While not conclusive, it would seem that a virus plays some part in the production of papillomas, and perhaps this is altered by hormonal changes.

Grossly, the lesions appear as grayish-pink, mulberry-like nodules projecting from the mucosa, varying in size from a few millimeters in diameter to lesions that completely fill the larynx. They may occur anywhere in the larynx, trachea, bronchi, epiglottis or pharyngeal wall but are most commonly found on the true cords and ventricular bands. They are friable and bleed easily when touched.

Microscopically, they are seen to be benign, sessile or pedunculated lesions with a vascular connective tissue core covered by proliferated stratified squamous epithelium. They arise from the mucous membrane and show no tendency to infiltrate below the basement membrane. The cells show frequent mitotic figures indicating rapid growth; however, the cells are all well differentiated and mature.

The first symptom of papilloma of the larynx is usually a change in character of the voice, which is soon followed by a hoarseness which may ultimately result in aphonia. The first respiratory symptom is usually a cough. This is followed by progressive dyspnea which, if untreated, may result in asphyxia. A change in character of the child to an inactive, retiring type is not infrequent.[1] As the disease progresses. one notes the increasing use of the accessory muscles of respiration, retraction of the supra- and infrasternal notches, and cyanosis.

Laryngeal papilloma in children is often a self-limited disease, the lesions disappearing at puberty. Smith[8] and Jackson[9] have reported cases that have undergone malignant degeneration, but all of these cases have been in adults.

If the papillomas fail to disappear at puberty, they usually take on the clinical characteristics of the adult type in that they show less tendency to recur and are often cured by simple surgical excision. A notable exception to this is a case reported by LeJeune,[10] in which the papilloma recurred in spite of 97 operative procedures and resulted fatally with a squamous cell carcinoma of the larynx.

The diagnosis is made by direct laryngoscopy, biopsy and microscopic examination of the tissue.

When confronted with a child who has chronic progressive hoarseness, one must, in addition to papilloma, consider other benign tumors, vocal nodules, "screamers' nodes," chronic laryngitis, congential cysts and webs, congenital laryngeal stridor, laryngotracheitis, hypertrophy of the ventricular bands, foreign body, paralysis of the recurrent laryngeal nerve, syphilis or tuberculosis.

The multiplicity of therapeutic procedures for multiple papilloma is evidence in itself of the failure of all of them to produce consistent results.

In regard to surgical procedures, it should always be borne in mind that in children the disease is often self-limited; therefore, nothing should be done that will result in any permanent deformity of the larynx. Gentle forceps removal under direct laryngoscopy is the most often practiced and is perhaps the best surgical procedure. This, at times, may be augmented by tracheotomy to obtain an adequate airway and may also be of value by placing the larynx at rest, as suggested by a case reported by Patterson,[11] in which recovery occurred following a tracheotomy only.

Numerous medications have been applied topically with varying and inconsistent success. Of interest is Broyles'[12] report of five cases treated by topical application of estrogens, amniotin in oil, in 1 cc. (10,000 U.) doses following surgical removal, in which a cure was obtained in all cases; however, Ferguson and Scott[2] were unable to duplicate these results with this therapy.

Varying results have been reported from the use of irradiation, both X-ray and radium. Of the two, external irradiation by X-rays seems preferable because of the lessened danger of perichondritis and the resultant permanent damage to the larynx;[13] however, with X-ray therapy the danger of producing permanent damage to the larynx still exists and is the chief contraindication to this method of therapy which, at times, seems to result in a cure.

Holinger[7] has recently given a preliminary report of seven cases of multiple papilloma in children treated with aureomycin in doses of 25 to 50 mgm. per kilogram per day following forceps removal. Two of the seven cases were apparent failures, but this may be the result of inadequate dosage of aureomycin. The remaining cases were all markedly improved. While it is yet too early to draw any definite conclusions, this form of therapy is rational and encouraging and deserves further trial.

The following is a report of four cases treated with terramycin* following forceps removal:

Case 1: F. J.,† a three-year-old colored male, who first came to our clinic on Sept. 6, 1950, with chief complaints of hoarseness and shortness of breath.

His illness apparently began when he was 19 months old, with an onset of hoarseness which gradually grew worse. In February, 1950, he was taken to another hospital in New Orleans because of this hoarseness, and a laryngoscopy was done, following which the child had more hoarseness and the mother refused to return with the child for further treatment. He was not seen again until he came to our clinic with a history of increasing respiratory distress of two weeks' duration and a marked increase in hoarseness.

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*"Terrabon," elixir of terramycin, was used in the three children; 250 mgm. capsules of terramycin were used in the adult. Both were generously supplied by the Pfizer Co.

†From the service of Dr. Carl Granberry and Dr. Adrain Cairns at the Eye, Ear, Nose and Throat Hospital of New Orleans.

Past history revealed that he had frequent upper respiratory infections as an infant. He had measles at the age of two years.

Family history was negative for hoarseness, syphilis, tuberculosis or other pertinent findings.

Physical examination on admission revealed a child who could speak only in a whisper and who was in marked respiratory distress, using all the accessory muscles of respiration and revealing marked abdominal and suprasternal notch retraction on inspiration

The patient was admitted to the hospital on Sept 6, 1950, and placed in an oxygen tent. The following day, under local anesthesia, a low tracheotomy was done, the cannula being inserted between the third and fourth tracheal rings. Five days later, he was given an ether anesthetic and, under suspension laryngoscopy, it was seen that the entire larynx and supraglottic region was filled with papillomatous tissue No visible airway was present. By gentle use of the forceps the papilloma was removed as far as possible without injuring the cords On the sixth postoperative day, he was started on terramycin, 50 mg *per* kg per day in four divided doses (one teaspoonful of terrabon every six hours) This was continued for 37 days, and four days after cessation of treatment he was re-examined under suspension laryngoscopy and the larynx was found to be perfectly normal.

On Dec. 5, 1950, six weeks after the previous examination, he was again examined under suspension laryngoscopy and there was seen to be a recurrence of papillomatous tissue which occupied approximately the anterior one-third of the glottic silhouette This was gently removed by use of the forceps, and the patient was again put on terramycin in the same dosages as before and continued for 30 days

On Jan. 16, 1951, he was again examined under suspension laryngoscopy and found to still have a small amount of papillomatous tissue at the anterior commissure. This was removed with a Mosher forceps The terramycin was discontinued and 10 weeks later on March 27, 1951, he was again examined with the suspension apparatus and multiple papilloma were removed from the right vocal cord in the posterior one-third and at the anterior commissure At this writing he has again been put on terramycin in a dosage of 250 mg every six hours and will be kept on this for a minimum of two months

Case 2: P. L. G.,* a six year-old white female whom I saw for the first time on Jan. 4, 1061.

Her chief complaint was hoarseness and shortness of breath Her illness was noticed when she first learned to talk at the age of 18 months. Her voice was weak and she was able to speak only in a hoarse whisper Her voice gradually became even worse and in February, 1947, two and one-half years after the onset, she began having progressively severe respiratory difficulty. On June 30, 1949, because of her respiratory difficulty, she was taken to another hospital where under suspension laryngoscopy, multiple papilloma were removed from the larynx Following this, she was apparently normal for two to three months, after which she again developed hoarseness and dyspnea, necessitating a second suspension laryngoscopy for removal of multiple papilloma This was done on

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*From the service of Dr. Kots Allen and Dr Mercer Lynch at the Eye, Ear, Nose and Throat Hospital, New Orleans La.

Feb. 9, 1950. On March 5, 1950, May 5, 1950, July 19, 1950 and on Oct. 6, 1950, she had papilloma removed from her larynx under suspension laryngoscopy, all procedures being mandatory because of respiratory difficulty.

Past history and family history were essentially negative.

On Jan. 4, 1951, she was admitted to the Eye, Ear, Nose and Throat Hospital in marked respiratory distress. She was taken to the operating room for suspension laryngoscopy; however, during the induction of anesthesia with vinyl ether, she vomited and became markedly cyanotic. A tracheotomy was immediately done and the patient promptly responded. The anesthesia was then continued and the larynx exposed with the suspension apparatus revealing the entire glottic chink to be obscured by the papilloma. The papilloma were removed so far as was possible without damaging the vocal cords, and two days later she was started on terramycin, 250 mgm. per kg. per day (250 mgm. every six hours).

On Feb. 2, 1951, she was examined with the laryngeal mirror and found still to have some papillomatous growth. The terramycin was continued in the same dosage and on March 21, 1951, she was admitted to the hospital and her larynx examined under suspension laryngoscopy was found to be free of papilloma. Terramycin will be continued for at least another month, after which time she shall return for re-examination.

Case 3: T. J.,* a 28-year-old colored female, who in 1945 noted for the first time the gradual onset of hoarseness. The hoarseness was progressive; however, there was no dyspnea. On June 26, 1945, she had multiple papilloma removed from her larynx at another hospital. She was improved for about one week, following which the hoarsness became progressively worse, so that by August, 1945, she was unable to speak. On Aug. 7, 1945, she again had papilloma removed from her larynx. Following this, she was improved; however, her voice did not return to normal. In June, 1949, her voice again began to get progressively worse, but she received no treatment until she came to our clinic for the first time on Dec. 28, 1950, where examination revealed multiple papilloma on both cords and subglottically. On Jan. 10, 1951, under suspension laryngoscopy, the papilloma were removed with Mosher forceps and the following day she was started on terramycin in a dosage of 50 mgm. per kg. per day in four divided doses.

Past and family histories were essentially negative.

She has been followed at weekly intervals and one could note the gradual disappearance of the small amounts of papillomatous tissue that remained after surgery. On March 3, 1951, the larynx was free of papilloma, although the edges of the cords were still slightly irregular. This was attributed to the three previous procedures she has had on her larynx. At this writing, she is still on terramycin in the same dosage and will be continued on this for at least another month.

Case 4: P. D. C.,† a four and one-half-year-old white male, with the chief complaint of dyspnea and hoarseness.

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*From the service of Dr. Kotz Allen and Dr. Mercer Lynch at the Eye, Ear, Nose and Throat Hospital of New Orleans, La.

†From the service of Dr. Kotz Allen and Dr. Mercer Lynch at the Eye, Ear, Nose and Throat Hospital of New Orleans, La.

His illness began at about the age of two years with the gradual onset of hoarseness, followed in several months by the development of respiratory difficulty which gradually became worse. On March 19, 1949, he was taken to another hospital where, under suspension laryngoscopy, multiple papilloma were removed from both cords, the arytenoids and the subglottic tissues. Following this, his voice began to get worse again, and in March, 1950, he again developed dyspnea, necessitating a second operation on March 10, 1950. On May 19, 1950, and again on Sept. 15, 1950, he required suspension laryngoscopy and removal of papilloma because of hoarseness and dyspnea.

Past and family histories revealed no significant findings.

On Jan. 3, 1951, he was admitted to the Eye, Ear, Nose and Throat Hospital where, under suspension laryngoscopy, multiple papilloma were removed from both vocal cords, the rest of the larynx appearing normal

On Jan. 14, 1951, he was started on terramycin in a dosage of 50 mg. per kg. per day in four divided doses Two months later, on March 16, 1951, he was readmitted to the hospital and the larynx examined under suspension laryngoscopy. He was found to have a small amount of papillomatous tissue at the anterior commissure, which was removed with Mosher forceps. The rest of the cords and the larynx were free of papilloma. At this writing, he is still on terramycin in the same dosage and will return for re-examination in one month.

In none of the cases was there any gastrointestinal disorders during treatment with terramycin. Complete blood counts were seen on all patients at the end of two months' therapy and all were within normal limits.

SUMMARY.

The incidence, etiology, pathology, symptomatology and treatment of multiple papilloma of the larynx are reviewed and discussed. Four cases of multiple papilloma of the larynx treated by surgery followed by terramycin are reported. Two cases are free of papilloma after two months of treatment. Two cases are improved but still have papilloma.

Complete blood counts on all the patients after two months of treatment with terramycin were within normal limits.

CONCLUSIONS.

Four cases are not enough on which to base any dogmatic statements, and not enough time has elapsed even to prophesy what the eventual outcome will be in these four cases; however, I feel that there is sufficient evidence on which to base several general statements.

Terramycin in relatively large doses, given after surgery, appears to have some beneficial effects in the treatment of multiple papilloma. In a dosage of 50 mg./kg./day, it seems necessary to give it for a period of not less than two months. The apparent effect of terramycin on multiple papilloma lends added support to the theory of a virus etiology of multiple papilloma.

Terramycin in the dosage used is apparently nontoxic, there having been no gastrointestinal disorders or significant changes in the blood picture of any of the cases.

Case 2 well illustrates the danger of attempting to give a general anesthetic to an individual with respiratory obstruction. All patients with respiratory obstruction should first have a low tracheotomy done under local anesthesia. This was not done in Case 2 only because of the parents' objections to tracheotomy.

Case 3, although an adult, was included in this study because of its unusual similarity to the childhood form of multiple papilloma.

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OTITIS EXTERNA IN PUERTO RICO.*†

A CLINICAL AND BACTERIOLOGICAL STUDY OF 82 CASES.

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The infections of the external ear canal constitute a considerable part of the routine ear, nose and throat office practice. They have been estimated to comprise about 5 to 20 per cent of all such practice, depending upon the season of the year. While the condition is frequently seen in tropical and subtropical countries during all seasons, still, both in these countries and in the temperate zones, it is more prevalent during the Summer.

Many synonyms have been applied to this disease: otomycosis, fungus ear, mildew ear, Panama ear, Singapore ear, swimming ear, itching ear, adobe ear, Dhobie ear, jungle rot, stink ear, Hong Kong ear and hot weather ear. Clinically, it is mainly an illness of tropical and subtropical countries, characterized by itching, swelling, pain, a sensation of fullness in the ear, discharge, deafness and, at times, fever and trismus. It may be an acute process or a chronic condition of many months' duration. In the more acute cases, more of the above signs and symptoms are present at one and the same time.

Gill[1] has pointed out as predisposing factors the following conditions: maceration of the canal, traumatism due to foreign bodies, climatic influences and individual susceptibility.

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Gordon,[2] who made his observations in the island of Guam, thought that climatic conditions and a low level of personal hygiene were the most predisposing factors. According to Simonton,[3] atmospheric humidity was the primary factor in 841 cases studied in New Guinea and the Philippines. This author places cerumen as the ranking predisposing cause, and believes swimming and bathing to be of secondary importance only. Mac Cormack and Martin[4] believe that there may be a relationship between seborrheic dermatitis of the scalp and the otitis externa.

Until some 20 years ago the general opinion on the cause of this disease was that it was due to the action of fungi. This was primarily due to the influence of reports in the older literature, mostly from the tropics. Thus, Graham,[5] working in Syria in 1890, obtained mucor corymbifer from the auditory canal of a Moslem woman suffering from intense pain in the ear, itching and tinnitus; several forms of aspergilli were isolated at the same time.

In the more recent literature, some authors have continued to stress fungi as causative organisms. Simms[6] reported three cases in 1937 that he thought were due to aspergilli. Minchew, Collins and Harris[7] found, in 1940, no essential difference between the bacterial flora of the normal and the infected ear canal, but found 27 to 30 per cent of 93 suspected cases of otomycosis, to have aspergilli in their ears, while these fungi were relatively rare in normal ears. Barber *et al.*,[8] in 1931, reported pityrosporon as one of the commonest causes of the condition and thought that the staphylococcus aureus was responsible for the production of boils, and streptococci for fissures of the canal.

On the other hand, the opinion has been growing to the effect that the principal causative agents of otitis externa are bacteria, especially those of the pseudomonas aeruginosa (bacillus pyocyaneus) group and certain streptococci. In 1932, Hermitte[9] reported 56 cases of external otitis, refractory to all forms of treatment, in 21 of which he obtained pseudomonas, and brought about a cure by means of an autovaccine.

In 1934, Bettington[10] reported a cure of single case of pseudomonas otitis, also by autovaccine. Working in Egypt in 1938, Morley[11] reported 100 cases of the condition, in a certain number of which the cause again appears to have been pseudomonas aeruginosa. Other authors who found pseudomonas as the only or principal invader in cases of otitis were Greaves,[12] Dunlap,[13] Quayle,[14] Simon,[15] Beach and Hamilton,[16] Jones,[17] Senturia,[18] Syverton *et al.*,[19] Salvin and Lewis,[20] Clark[21] and Castle *et al.*[22]

Mitchell[23] and Ponce de León[24] stressed the importance of streptococci as pathogenic agents, an opinion that was much strengthened by the careful bacteriological work of Williams, Montgomery and Powell,[25] in 1939.

Other bacterial agents have been incriminated to a certain extent. For example, Salvin and Lewis[20] encountered, among 100 cases of external otitis, 27 with staphylococcus albus, 14 with various diphtheroids and nine with streptococcus viridans. Simon[15] isolated, in 90 patients, staphylococci from 20 per cent, bacillus proteus from 15 per cent, bacillus coli from 15 per cent, and diphtheroids from 6 per cent; however, in both of the two last mentioned studies, pseudomonas aeruginosa was the organism most frequently encountered in both pure and mixed infections.

In view of the importance of otitis externa in otorhinologic practice, of the fact that the opinion still prevails in certain sectors of the profession as to its fungous cause, and because of the disposal of sufficient and adequate clinical material for study, we decided to investigate the condition from the etiologic and therapeutic points of view.

METHOD OF STUDY.

A total of 82 out-patients in different stages of otitis externa, both acute and chronic, were selected. Material was taken from 112 ears, by carefully swabbing the external ear canal for use in the bacteriological studies, and then scraping gently with sterile curettes to obtain material for the mycological work. These samples were immediately sent to the bacteriologist and mycologist, respectively.

The results of this study may be briefly and conveniently summarized as they appear in Table 1.

TABLE 1.

Organism	Pure Infections (No of Cases)	Mixed and Pure Infections (No of Cases)	Per Cent
Staphylococci	19	42	36.1
Ps. aeruginosa	9	21	18.1
Diphtheroids	7	23	19.8
Aspergilli	4	10	8.6
Streptococci	0	10	8.6
N. catarrhalis	3	5	4.3
B. coli	2	4	3.4
Penicillium	1	1	0.86

It is clearly seen that fungi play a negligible rôle, at least in the community in which the study was conducted, and that the most important organisms causing otitis externa are staphylococci, in the first place, and pseudomonas aeruginosa, in the second. The largest number of pure infections was 19, all of them due to staphylococci, which also predominated among the mixed infections. While pseudomonas was slightly more frequently encountered (nine cases as a pure infection than the diphtheroids (seven cases) the total number of times that the latter were isolated was only slightly larger than that of pseudomonas; however, we attach no pathogenetic significance to the presence of diphtheroids in the external ear canal.

As regards mixed infections, we found that the largest number in which a certain organism appeared in combination with one or more was six cases, and these were staphylococci with diphtheroids and staphylococci with pseudomonas. It will be noted that both were *examples of combinations* with staphylococci, but since the total number of cases was only six for each combination of organisms, it is seen that this is not of statistical importance.

The staphylococci isolated were *albus* in 25 cases and *aureus* in an equal number. They gave rise to no clinically detectable differences.

The results of cultures were of great clinical significance both as to acuteness or chronicity, and as to the results of treatment, as discussed above in that section of the paper.

CONCLUSIONS.

1. A bacteriologic, mycologic and therapeutic study was conducted on 82 cases of otitis externa.
2. Fungi were found to be of very minor etiologic importance.
3. In most cases, staphylococci were isolated in pure or mixed cultures, and these were mostly acute infections.
4. In the second largest group, pseudomonas aeruginosa (bacillus pyocyaneus) was present, and these were all chronic.
5. Treatment by simple cleansing, with or without the instillation of sulfanilamide or sulfathiazole powder, is curative in most cases, within 12 days, when the cultures have yielded Gram positive organisms.
6. Simple cleansing of the ear canal, followed by the instillation of weak acetic acid solution, yields good results when pseudomonas is present in the cultures, but not in less than two to eight weeks.

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PEARLY TUMOR OF THE CEREBELLOPONTINE ANGLE. REPORT OF A CASE WITH REVERSIBLE DEAFNESS.

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Pearly tumors of the cerebellopontine angle comprise only about 6 per cent of all tumors occurring in this region. Because of their characteristic mother-of-pearl appearance, they have been called "pearly tumors." Also known as cholesteatomas and epidermoids, they are benign, encapsulated, congenital neoplasms which arise from aberrent epidermal inclusions.

Unlike other tumors of the cerebellopontine angle, symptoms and signs referable to the VIIIth cranial nerve are unusual; of 13 cases reported by Gonzalez Revilla,[1] 10 patients had normal hearing and nine normal labyrinths. Olivecrona[2] reported normal VIIIth nerves in five of seven patients with pearly tumors.

The commonest symptom produced by pearly tumors of the cerebellopontine angle is pain referable to the Vth cranial nerve. It most frequently involves the third division and may mimic tic douloureux.[3,4] According to Olivecroma,[2] the typical manifestations of these neoplasms are: *1.* typical tic pain beginning in the third division of the trigeminal nerve in a young person; *2.* normal Roentgenogram of the skull; *3.* normal results of neurologic examination; and *4.* dislocation of the cisterna pontis and cisterna ambiens in the encephalogram.

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The following case is of interest for two reasons: first, return of hearing following removal of a cerebellopontine angle tumor is rare,[5-7] and adequate preoperative and postoperative records of hearing and labyrinthine function are even rarer; second, it seems worthwhile to emphasize again that such a lesion can mimic tic douloureux. The explanation for disappearance of the pain for more than 10 months after alcoholic injection of the mandibular nerve is admittedly obscure.

REPORT OF CASE.

R. P. Y., a white man aged 28 years, was first seen by one of us (D. H. E.) on Oct. 31, 1949, because of pain in the right side of the face, progressive deafness and tinnitus on the right side, all of three years' duration. The pain, which was sharp, lancinating and of brief duration, was located at first in the upper gums and teeth, but later extended to the lower jaw. In the beginning, attacks of pain occurred many times daily, but later there were remissions for as long as six weeks.

A brief examination revealed no abnormalities except a slight decrease in hearing on the right to watch and voice tests. Audiometric and labyrinthine studies were not done.

The diagnosis of tic douloureux was made. Complete relief of pain was obtained after alcoholic injection of the mandibular division of the right Vth nerve on Nov. 9, 1949.

The patient had no further difficulty until Sept. 26, 1950, when he noted mild pain in the right upper teeth and beginning disappearance of the numbness. Because of this pain he returned to us Oct. 6, 1950. The pain in the second division of the Vth nerve on the right side became severe, the deafness on the right became progressively worse, and slight nystagmus on looking to the right developed.

A presumptive diagnosis of a lesion of the right cerebellopontine angle was made. Otorhinolaryngologic examination revealed total deafness on the right, mild nerve deafness on the left, dead labyrinth on the right (no response to 30 cc. ice water) and a normal left labyrinth. The blood glucose level was elevated and there were 15 cells per cmm. in the cerebrospinal fluid. A Roentgenogram of the skull showed no abnormalities.

On Nov. 10, 1950, unilateral suboccipital craniotomy revealed a pearly looking tumor in the right cerebellopontine angle. One centimeter of the VIIIth nerve was seen between the tumor and the internal auditory meatus. The tumor concealed the Vth cranial nerve and displaced the IXth, Xth and XIth nerves caudally. After bloodless piecemeal removal, the compressed and displaced pons and the greatly elongated and flattened Vth nerve came into full view.

The patient returned to work on the twenty-fourth postoperative day. Since operation there has been a progressive return of hearing (see Table 1) and labyrinthine function (see Table 2).

TABLE 1. AUDIOGRAM (DECIBEL LOSS).

Cycles/Sec.	256	512	1024	2048	4096
10-25-50	No hearing with masking				
11-20-50	60	60	60	60	60
6-30-51	30	20	20	10	80

TABLE 2. CALORIC TEST, RIGHT EAR.

10-27-50	No response to stimulation with 30 cc. ice water.
6-30-51	Normal response to stimulation with 5 cc. ice water.

SUMMARY.

A case of pearly tumor of the cerebellopontine angle simulating tic douloureux is reported. It is of particular interest because return of hearing followed removal of the tumor.

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Audicon Models 400 and 415.

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Silvertone Model J-92.

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THE
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NUTRITION AND THE EYE, EAR, NOSE AND THROAT.

(With Excerpts from the Literature.)

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This is the tenth editorial on this subject, and our annual comment has paralleled the general trend in Nutrition. At first vitamins monopolized the attention; we noted that their discovery sparked an awakened interest in foods and their direct bearing on health. In those days the literature on vitamins was so voluminous that it constituted a considerable portion of current medical literature. Of course, all nutrition studies are the concern of the physician regardless of his specialty, but much of this early literature dealt directly with the Eye, Ear, Nose and Throat.

Today the situation is very different — in contrast to only a decade ago. Now in order to uncover bits of news important to our specialties one has to search in strange and sundry places. They are certainly not to be found in Eye, Ear, Nose and Throat literature.

VITAMINS.

There is now a wealth of information about the vitamins present in food — but not of the vitamins created within the

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body by the intestinal bacteria. This is obviously of great importance in nutrition; also, anyone who prescribes the sulfa drugs or antibiotics is deeply concerned with their action on these bacteria. Although the behavior and meaning of intestinal flora are far from clear and the solution of this complex problem lies in the future, yet we can be guided by certain facts already available.

There seems to be no doubt that synthesis of vitamins has been proved in animals, and the general concept is that the same process occurs in man — although some consider that definite proof is lacking.

Najjar[1] expressed his opinion in 1945, after experiments on man as well as animals, and his further studies confirmed the former findings. His concept is given as follows: To a greater degree than generally realized vitamins are produced within the body. Production of vitamins occurs in the tissues themselves or within the bowel. Animals which do not require "C" in their food appear to synthesize it in their tissues. A simple example of countless observations is in the synthesis of "B_2" which is found in the milk in ten times the amount ingested by the cow. The synthesis of "B_2" also occurs in the feces of fowl, particularly after their passage from the bowel. When the sulfa drugs appeared it was found that they interfere with this intestinal synthesis of vitamins and it then became possible to study not only the separate vitamin requirements but also the extent of the synthesis. Merely suppressing such synthesis caused deficiencies that could not only be observed but also measured. Of great importance in man is the production of the B Complex by bacteria in the intestine. Man can synthesize "B_1", "B_2", nicotinic acid, biotin and folic acid. The ability to do this presents a new approach and a different outlook on man's food requirements. Obviously the amount of a vitamin in food is less than the physiologic need for it; the difference is accounted for by the amount synthesized. Clinically, the length of time sulfa drugs are ordinarily taken can hardly suffice to precipitate a state of vitamin deficiency; but the possibility of inhibiting synthesis long enough to have a deleterious effect should certainly not be ignored.

The experiments of Daft and Sebrell[2] indicate that "K" also is synthesized in the bowel. They comment that hemorrhage in the newborn, preventable by giving "K", occurs when the bacterial flora of the bowel has not yet become established; and that, later, intestinal synthesis of "K" ends this period of danger.

This problem, however, is far from simple. Recent experiments on the growth of chicks and poults[3-5] suggest that antibiotics actually help in the production of *more* vitamins — possibly by inhibiting some bacteria and aiding others. The poults grow faster and larger. A current concept is that certain bacteria utilize ingested vitamins; that these bacteria may be inhibited by the antibiotic; and so these vitamins remain fully available to the host.

ANTIBIOTICS.

It is only recently that two new side-effects have come to light. These are of direct importance to anyone who prescribes antibiotics. The time is not yet when it can be stated that antibiotics 1. lessen the supply of vitamins and 2. also permit yeasts and molds to flourish. However, among the limited number who have so far considered this matter, there is a trend of opinion in that direction.

Even in the very few years since the advent of antibiotics — it was only ten years ago that penicillin was introduced — the results have been not only good but startling. With this truly new epoch, first of the sulfa drugs and then of the antibiotics, the practice of medicine and surgery has been markedly changed. Occasionally certain unfavorable and even serious results have been noted, but from the standpoint of statistics and percentages the experience has been most favorable. By and large, the toxic reactions are not sufficiently serious to require that the antibiotic be stopped. The good far outweighs the bad. It is of course to be expected that any such powerful therapy would have some bad side-effects.

Deaths have occurred. To be sure, bad effects do not occur very often but they are caused by the antibiotic just the same.

Unfortunately, the patient who "just feels bad" confidently expects a "wonder drug" and a quick cure. This attitude has prevailed ever since the advent of penicillin — in fact even before that, when the sulfa drugs were introduced.

Our personal experience with bad side-effects has been *mostly with streptomycin. We conferred with Dr. John S.* Chase[9] on a large series of Veterans and it seemed to us that the streptomycin had attacked the Central Vestibular Tract,[10] as was the case in a physician whom we had studied. After taking streptomycin for a mild bladder infection he was seriously incapacitated for over a year and never fully recovered. This doctor, a neurologist, had the rare opportunity to study himself as a guinea pig. His hearing was normal. He had no vertigo. When he turned over in bed to his great surprise he kept on rolling. When he sat up in bed he kept on going and his head landed at the foot of the bed. "The brakes didn't work." When he gazed at an object across the room he could see it clearly but when he turned his head quickly the object jumped out of sight and he had to hunt for it. When he could attempt to walk he was unable to balance and reeled — in no particular direction. When he was able to ride in a wheelchair he could not tell with the eyes closed when a corner was turned. The nurse rotated him in the wheel-chair but he had no sensation that he was turning. In brief, he had lost automatic ability to keep right side up, to maintain equilibrium, to recognize movement with eyes closed and to maintain fixation of gaze when the head was moved. After a year he prided himself on being able to put on his trousers standing in the middle of the room. By continual practice he has compensated considerably for his vestibular loss.

In spite of the remarkable benefits of streptomycin, especially in tuberculosis, it is apparent that like any antibiotic this rose has its thorn.

It already seems clear that after antibiotics are given monilia may flourish; and that they, normally present and inoffensive, seem to become pathogenic. Although there is as yet no explanation, the fact is that one may see oral lesions —

monilia by culture — promptly after giving an antibiotic. Also for some unknown reason, giving B Complex has a good effect — in both prevention and treatment. At the moment these might be termed "mysterious facts."

It seems strange that the concepts of a possible B Complex deficiency and the presence of lesions due to monilia have not been noted and reported in the past; it was less than two years ago that Harris[11] made the first comprehensive report. The possible deficiency and the definite lesions from monilia might well be termed "the two new Cautions" in the use of antibiotics. Our impression of this matter is that we should not make too much of it — or too little; but we should certainly be alert.

Although time may alter present concepts as new facts come to light, enough evidence is now available, as given in the following excerpts, to indicate that *1.* Physicians, especially otolaryngologists, should be watchful and aware of these two new "Cautions"; *2.* We should certainly be more hesitant in giving antibiotics so freely, especially for trivial ailments; and *3.* It would seem advisable to give the B Complex *with* the antibiotic especially when it is given over a long period. The B Complex itself can be given and, in addition, supplemented by foods that ars rich in the B Complex, such as liver.

If, as it now seems, antibiotics may be followed by moniliasis whereas sulfa drugs are not — one wonders what the reason might be. Antibiotics and monilia have something in common; antibiotics are derived from fungi — each one from a different fungus (with the exception of two which are derived from bacteria).

* * * *

Aureomycin was given orally by Harris[11] to 135 and chloromycetin to 46 patients with brucellosis. Many developed lesions in the mouth — aften within two to three days — first on the uvula, then the hard palate, pharynx, cheek, lip and tongue. The coating of the tongue changed from yellow to brown and eventually black unless treatment was interrupted.

Resolution of all oral lesions was of variable slowness; in some patients they persisted for several weeks. When the B Complex was given to a later group of patients such lesions occurred less frequently or resolved more rapidly. Later, when larger parenteral doses of the B Complex were given daily together with the antibiotics, the mucous membrane lesions occurred in fewer patients and in lesser degree. Harris ponders on the meaning of these lesions, set into motion by antibiotics. He considers that monilia may over-grow and gather virulence following the antibiotic effects on the intestinal bacteria; or that monilia may invade tissues whose resistance may have been lowered by a B Complex deficiency. He suggests careful supervision during, and for long periods after treatment with antibiotics.

It was Harris who first presented this new concept; he now writes us[12] that eighteen months subsequent experience confirms his original impressions, as follows: Big doses of B Complex, preferably parenteral, given with the antibiotic largely prevent monilial infection of mucous membranes as well as the manifestations of an apparent B Complex deficiency. This adds weight to the assumption that there primarily is a B Complex deficiency, with subsequent overgrowth of fungi. Further, when B Complex is given every other day lesions may appear, but if the dose is then given daily all lesions subside, even though the antibiotic is continued. He reasons that perhaps it is not only the vitamin deficiency which permits the fungi to flourish; possibly the elimination of normal intestinal bacteria plays a rôle, in that these may be natural antagonists of the fungi. Although purely an assumption, it may be that the fungi become virulent. Monilial lesions are not necessarily self-limiting even if the antibiotics are stopped. While they usually subside spontaneously, some require fungicidal treatment. One patient had steadily progressing mucous membrane lesions (monilia) for four months after the antibiotic was stopped. There was then a prompt response to fungicidal therapy. The rapidity with which mucosal lesions develop suggests to Harris that they could hardly be due to vitamin deficiency; and yet when B

Complex is given parenterally the lesions rapidly clear up. Also, the lesions exactly resemble those usually produced by a B Complex deficiency.

The same side-effects as these given by Harris were noted by Gewin and Friou;[13] in addition they observed prothrombin deficiency, visual disturbances with retinal changes and severe peripheral neuritis. Over a period of months they most carefully studied a patient in whom aureomycin and chloramphenicol effected a bacteriologic cure of endocarditis. After severe intestinal bleeding the prothrombin was 43 per cent; r.b.c, 2,100,000; hb, 7.0 gm. The eye findings included yellow vision, blurring and fading of letters while reading, spots in front of the eyes, slight blurring of both optic discs, granular changes in the retina and enlarged blind spots. These side-effects were attributed to deficiencies in the B Complex and "K". Vitamin therapy proved only partially effective until the antibiotics were stopped.

Increasing experiences with antibiotics are revealing a commensurate increase in toxic side-effects. It seems to Woods, Manning and Patterson[14] that one group of untoward effects is particularly deserving of emphasis and investigation. When bacteria are exposed to antibiotics, those susceptible are suppressed; but the non-susceptible may grow abundantly, even in the presence of high concentrations of the antibiotic. Much has been learned concerning bacteria but little concerning fungi; also there is scant information as to the effect of this suppression of bacteria on coexisting fungi. The authors encountered many cases of clinical moniliasis, in most of which the condition was apparently a direct sequel of antibiotic therapy. The first group of patients had sore mouth, hairy tongue, perleche — the clinical picture of thrush — resulting from the local use of penicillin or aureomycin as sprays, troches, soluble tablets or powders. These preparations have been widely prescribed by physicians and druggists and these reactions have been so frequent as to cause many physicians to discontinue their use. Monilia were obtained from the mouths of all. In a smaller group which developed diarrhea monilia were abundant in the stools. Of 25 patients the mouth and throat were involved in 20. The sequence was sore throat, antibiotics (local or systemic), recovery; then after one to three days the oral and upper respiratory lesions already described. The tongue is most frequently involved and at first is thickly coated, often with an over-growth of the papillae, giving the appearance of a brown or black hairy tongue. Later the coating or hairy covering comes off and the tongue is left beefy red, dry and swollen with papillae standing out in relief. It is tender to palpation and on contact with acid juices and foods. The sense of taste is markedly altered. The buccal mucous membrane is at first covered with whitish exudate which can be wiped away easily. As this comes off it leaves the mucosa glazed and parched. Less often this exudate is seen on the tongue, soft palate, tonsils and pharynx. There are often small petechial hemorrhages in the mucosa. The palate, uvula, aryepiglottic folds and pryiform fossae are acutely red and edematous. In one case the exudate extended the entire length of the esophagus.

The chance of getting a deficiency on a good diet seems to Jukes[15] to be small, because we do not know of any way in which antibiotics would interfere with the assimilation of vitamins normally present in the food. The only change which might be produced by antibiotics would be to lessen production of vitamins by intestinal bacteria. Jukes considers that even this deficiency would not be noticed because of the vitamins provided by the food, in a preformed state. He further comments on the oral lesions occurring after a few days local use of antibiotics and considers that such deficiency could hardly be produced in so short a time; he regards such lesions as due to a local reaction to the antibiotic. As to monilia, he suggests that by eliminating bacteria an antibiotic may render available to yeasts and molds the nutrients otherwise appropriated by the bacteria.

Leitner[16] also states that it takes considerable time before definite deficiency symptoms and signs are produced experimentally in well-nourished healthy subjects. In spite of this recognized fact he reports a patient who, after aureomycin, showed gross angular stomatitis and cheilosis, painful tongue

— shiny, swollen and denuded. Large doses of the B Complex healed the cheilosis and stomatitis in three days; the tongue became painless though still swollen. It would be tempting to speculate, says Leitner, if such an acute deficiency is due to interference with biosynthesis — or to "anti-B" vitamins evoked by the antibiotic.

Impressed with the work of Harris, Tomaszewski[17] undertook a careful study of these two new side-effects of antibiotics. They occur most frequently following chloramphenicol and aureomycin. They are usually not of a serious nature. As the use of these two antibiotics is rapidly increasing, a study of the side-effects is important. There is a dramatic disappearance of bacteria, and the appearance of fungi — usually monilia. Observations were made on 126 cases. In each case bacteriologic tests first showed that the pathogenic organism was sensitive to both of these antibiotics. The tongue was scraped with a sterile slide every two or three days in all cases before, during and for one to three weeks after cessation of treatment. Cultures were made for identification of fungi and bacteria. Both antibiotics were given by mouth in capsules. The side-effects were seen more often in patients treated for a longer time and with large doses. In some cases diarrhea occurred shortly after the starting of treatment; more often the patients complained only of loose stools. In a large proportion of patients the stool became odorless after a few days and in some this continued for a time after the cessation of treatment. Several had lesions of the rectum, anus and vagina. The most frequent lesions, however, were oral. Dryness of the mouth was noted — similar to that produced by atropine; the amount of saliva was reduced. Changes of the tongue developed in the majority of cases. Usually after two or three days there was a gradual disappearance of the normal whitish coating and a denuding of the tongue. Later a fiery red, raw, beefy tongue with partial atrophy of the papillae was often seen. The lingual manifestations appeared more readily if the patient had been treated previously with penicillin or streptomycin. Soreness often started at the edges of the tongue. After stopping the antibiotic sometimes the tongue remained red and denuded for one, two or more weeks. In 10 per cent, some shortening of the papillae took place during the first few days' treatment and a brownish coloration appeared, gradually spreading over the surface. In four cases a true black hairy tongue developed. The sense of taste was often markedly affected. Redness of the mucous membrane of the mouth and throat often accompanied the changes in the tongue; also angular stomatitis. The changes in the mouth and on the lips were very similar to those seen in a deficiency of B Complex, especially riboflavin; so the vitamins were given and recovery seemed to proceed quickly, though not in a dramatic way. In some patients the B Complex was given together with the antibiotics; although the lesions were not prevented their intensity was definitely less. In most cases the bacteria disappeared in a few days after starting the antibiotic; at the same time fungi appeared in increasing numbers. In some cases monilia were found in large amount in the sputum, in the stool and in the urinary sediment. The normal bacterial flora usually return in one to two weeks after stopping the antibiotic, but fungi may persist for some days longer. The frequent coexistence of lesions in the three body orifices — mouth, anus and vagina — suggests some close relationship between them. The lesions may result from a chemical action on the local cells. At any rate absence of bacteria seems to permit the fungi to flourish.

In reporting a death from monilia Gausewitz, Jones and Worley[18] note that the patient had prolonged treatment with penicillin and then with chloromycetin. Autopsy showed multiple involvement of viscera by the monilia, by both histologic and bacteriologic study. They comment on the recent reports suggesting that antibiotics may act to increase the pathogenicity of monilia. Often monilia are isolated from healthy persons; they have been cultured from the lungs in nine of 71 routine autopsies. Stomatitis (thrush) is the most widely recognized manifestation; frequently the respiratory tract, vagina and skin are involved. Infections of various organs including the esophagus, bronchi and heart have caused death.

Fatal meningitis has been reported in four cases, in three of which monilia were identified by culture. In a meningitis patient who recovered monilia were seen in direct smears and identified in cultures of the spinal fluid.

* * * *

INTRAVENOUS FEEDING.

In our specialties intravenous feeding is rarely indicated but the internist and general surgeon have made advances in this field from which we can learn much. A book by Elman[19] concerns this subject only. At the turn of the century such procedures were limited to salt or glucose solutions. With advances in knowledge of nutrition parenteral feeding was devised for those who cannot eat, so that now all the six nutrients can be given by vein — water, salts (electrolytes), carbohydrates, fats, vitamins and proteins. Carbohydrate can be injected only when reduced to its basic form, glucose. Fats have been suitably emulsified although they are not yet available for general use; but mixtures of necessary amino acids are now available to furnish complete protein nourishment. Elman points out that a low level of "C' in the plasma does not necessarily signify clinical scurvy; this does result with the loss of "C" in the leukocytes. Determination of "C" in the plasma is relatively simple; but in the leukocyte it is more difficult and less standardized. This detection of "C" loss in the plasma permits treatment of scurvy in its "latent" phase, thus forestalling the onset of "active" scurvy. The need of "C" increases greatly after operation or injury. Large amounts of "C" are extravasated into the injured areas; it seems that the healing process requires much more "C" than is normally present in the tissues. This depletes the available "C". In extensive injuries such as burns, as much as 2 grams per day may be required to maintain a normal blood level, even though the total amount of "C" in the body probably does not exceed 5 grams. "B_1" is depleted by intravenous glucose and, like "B_1", riboflavin and niacin are also used up in carbohydrate metabolism. Other vitamins are sometimes given; there is no objection since no deleterious effects occur. Some have gone so far as to inject crude extract of liver because it may contain unknown factors important in restoring the patient to normal condition. Although "A" is fat-soluble it is available in water-soluble form as carotene. There is a simple chemical test for "A". After severe hemorrhage plenty of fluid remains in the body; 11 liters are in the interstitial spaces and much of this is mobilized quickly. Unfortunately this fluid contains very little protein, so that the addition of this fluid to the circulating blood results in both anemia and hypoproteinemia. If a soldier dies after a sudden loss of 2 liters of blood one explanation might be that death is due to lack of 60 grams of plasma protein — the amount lost. Then the inability of the body quickly to replace this would be a tragic example of "too little and too late". Not only are amino acids the basic elements of all protein but they circulate in the blood in which they maintain a constant concentration just as glucose does. Amino-acids-are-to-protein what glucose is to starch. Glucose is the actual fuel and amino acids are the building stones which replace the wear and tear of tissue.

If the day comes when glaucoma and edema of the internal ear are conquered, it will be through knowledge and control of osmosis. The electrolytes (*i.e.* salts) are distributed in three spaces in the body, as mentioned by Jones[20] — the cell space, the interstitial space and the vascular space. Body water makes up about 70 per cent of the body weight; of this 50 per cent is in the cell space, 15 per cent in the interstitial space and only 5 per cent in the vascular space. We see at once that only the smallest space, the vascular, is accessible to us for measurement and treatment. What is going on in the other larger spaces, although our prime interest — as in glaucoma or hydrops of the internal ear — is something we can judge only indirectly. Present teaching is that sodium salts are found in the interstitial and vascular spaces while potassium salts are found in the cell space. Water moves freely back and forth between the three spaces in response to changes in osmotic and circulatory pressures. The most important substances exerting osmotic pressure are sodium salts in the interstitial space, proteins in the vascular space and potassium salts in the cell space.

* * * *

"A".

There is little that is new about the well-known vitamins present in food. Of course water-soluble vitamins are freely eliminated whereas oil-soluble "A" and "D" are not. It has been known for years that overdosage of "D" can cause serious damage and many warnings have been issued. However, Caffey[21] now reports poisoning from excess of "A" — tender swellings, X-ray changes in the skeleton and increase of "A" in the blood. His patients received the usual concentrates of "A" and "D"; however, it seems that the "D" was not the toxic factor — because it has been used clinically in much higher doses without bad effects. Recovery from "A" poisoning is dramatically rapid — just from stopping the vitamin. Three types of concentrate commonly used were found to be toxic — if given in too large dosage. The hazards of such poisoning are considerably greater than the hazards of an "A" deficiency. Overdoing is worse than doing nothing.

A recent experience of Sulzberger and Lazar[22] concerns an adult. She had actually been taking 600,000 units of "A" daily for eighteen months. This self-medication was inspired by a radio commentator who recommended "A" as helpful in "keeping us from catching colds". Menses were shortened by three days. There were generalized joint and bone pains. Skin changes were the main feature — pigmentation, pruritus, dry rough skin with desquamation and excessive loss of hair including complete loss of eyebrows and lashes. The "A" was stopped at once; in addition, foods rich in "A" were restricted. Although vowing to cooperate she was reluctant to believe that anything as beneficial as a vitamin could actually cause trouble. She was convinced, however, when the joint and bone

The public response shows that there is a surprising interest in Nutrition. Certain of these ideas are presented here as being helpful to a patient.

In vacation time the average child selects foods of poor value and eats irregularly; too often by the end of the Summer his condition is worse rather than improved. There is more food value in a dollar's worth of milk and eggs than in a dollar's worth of pie and cake. The child should have natural foods, not processed foods; and particularly, not refined sugar or refined flour.

The patient often asks: "What's wrong with sugar?" A safe answer is: "Refinement and Overindulgence". Of course even refined sugar has a definite value; it is well and accurately known as a source of immediate energy; however it is not a nutrient; it cannot be converted into body tissue; it is just fuel.

In their natural state there are many good sugars such as the raw sugars — cane, beet, maple, honey, date and fruit. "White sugar" today, however, means a refined product from which vitamins and minerals have been removed. As such it is no longer a nutrient. It provides calories only.

As to over-indulgence, the use of "sweets" has increased until now they provide one-seventh of America's calories — as sugar or in soft drinks, candy, ice-cream and bakery products. They satisfy the appetite and supplant valuable and needed nutrients. Our ancestors had no refined sugar; in fact they ate long before food processing of any kind had become a profitable business. The use of these unnatural foods of civilization has led to malnutrition and denegerative disease.

It is hard for most people to look upon refined sugar as a serious threat; few have even given thought to the matter or realized the extent of its use. Education of the layman is not the only need; many physicians, including pediatricians, provide candy suckers for their little patients and naturally parents and children come to consider this as approval of sugar.

Of the four tastes — sweet, sour, salt and bitter — by far the most liked is sweet. If one feels he must have sweets, saccharin is available, now so purified that it has no bitter aftertaste. It is 500 times sweeter than sugar. As it is not a food its use has no effect except to provide sweetness.

To "see the dentist" is not the answer to the tooth-decay which is present in 95 per cent of the population. The dentist accomplishes most when he guides the patient in the selection of good foods. It is believed today that the chief cause of caries is eating refined carbohydrates. The gums and sockets also go bad on refined carbohydrates and the teeth loosen and fall out. With rare exceptions, when bone is lost around a tooth it is not replaced.

Sunshine acts on the oil in the skin; this results in the formation of "D". If this oil is all "soaped away" this will not occur. The United States occupies only 6 per cent of the earth's surface — but has 92 per cent of the world's bathtubs. So it is quite possible that we overdo the bathing.

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THE INNERVATION OF THE COCHLEA (GUINEA PIG).*

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INTRODUCTION.

Since the time of Retzius, extensive work has been done on the innervation of the cochlea. On the basis of the course taken by the fibres, two types of peripheral processes emerging from the bipolar cells in the ganglion of Corti are described by von Ebner[1] and by Lorente de Nó[2]: *a.* the radial fibres which go directly to the hair cells and *b.* the spiral fibres with extensive ramifications.

a. The radial fibres (see 1, in Fig. 1) run directly from the ganglion cells to the inner hair cells where each fibre innervates several neighboring cells and each cell may be innervated by several fibres. Lorente de Nó states that these fibres carry impulses of a very definite "local sign." According to the observations of Held,[3] the innervation of the inner hair cells is richer and more complicated than that of the outer hair cells. Many fibres meet at this level forming such compact innervation that the course of individual fibres is lost.

b. The spiral fibres are grouped in four bundles: *1.* intraganglionic spiral bundle; *2.* internal spiral bundle; *3.* tunnel spiral bundle; *4.* the external spiral bundle.

1. The intraganglionic spiral bundle (see G.B., in Fig. 1) runs apicalward inside the ganglion of Corti for distances, established by several authors, as much as one-quarter of a

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turn or more. Collaterals are constantly given off to the organ of Corti, and according to Lorente de Nó the collaterals innervate, almost exclusively, the inner hair cells (see 2, in Fig. 1). The work of Rasmussen[4,5] shows that the so-called superior

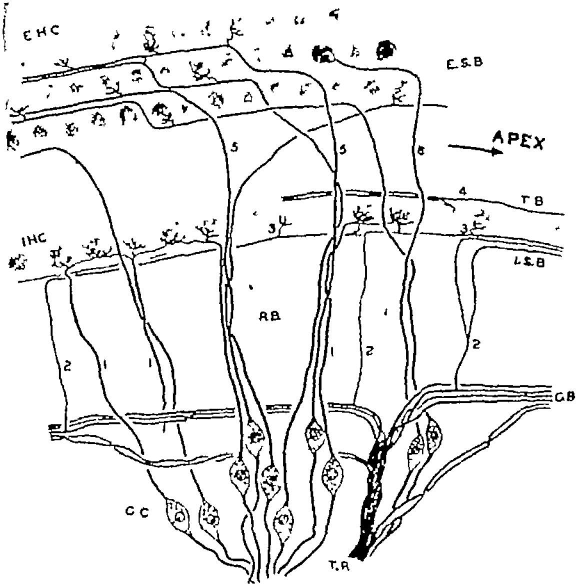

Fig. 1. The diagram represents the innervation of the cochlea based on observations of Lorente de Nó, Held and Rasmussen. E.H.C., outer hair cells. I.H.C. inner hair cells. G.C., ganglion of Corti. T.R., tract of Rasmussen which breaks up into several fasciculi which bend toward the apex and the round window to form the intraganglionic spiral bundle G.B., R.B. radial bundles formed by different types of fibres in their course inside the lamina spiralis. (1) Radial fibres to the inner hair cells. (2) Collaterals of the intraganglionic spiral bundle that later form the internal spiral bundle (I.S.B.) underneath the inner hair cells. (3) Collaterals from the internal spiral bundle to the inner hair cells. (4) Tunnel spiral bundle (T.B.), whose fibres turn back to innervate the inner hair cells. (5) Fibres to the outer hair cells that later form the external spiral bundle (E.S.B.) with multiple ramifications. (6) Fibre to the external hair cell unbranched.

olivary peduncle has its origin in the superior olivary complex and its terminals in the cochlea of the opposite side. The tract reaches the cochlea at the level of the first turn (see T.R., in Fig. 1) and enters the ganglion of Corti where it bends apicalward. He states also that morphologically the tract is formed by *efferent* fibres, a point of anatomical description which had been missed by all the previous workers in this field. From his observations[6] he presumed that the terminals of the tract could be identified with the intraganglionic spiral bundle. According to the observations of Lorente de Nó and others, the inner hair cells are innervated by radial·fibres and by collaterals of the intraganglionic spiral bundle. It may then be concluded that the inner hair cells have both an afferent and an efferent innervation.

2. The internal spiral bundle (see I.S.B., in Fig. 1) is formed by a group of fibres coursing beneath the inner hair cells. According to Lorente de Nó these fibres are the terminals of the intraganglionic spiral bundle. They give off numerous collaterals to the inner hair cells (see 3, in Fig. 1) and occasionally some fibres cross to the outer hair cells. He also states that these fibres run almost exclusively apicalward. According to Retzius, the fibres run either to the apex or to the round window.

3. The tunnel spiral bundle (see T.B., in Fig. 1) is formed by a small number of fibres running inside the tunnel. According to Held, they turn back to innervate the inner hair cells (see 4, in Fig. 1), but Wever[7] points out that it is possible that some of them cross the tunnel to the outer hair cells.

4. The external spiral bundle (see E.S.B., in Fig. 1) is formed by fibres coming directly from the habenula perforata. The fibres cross the tunnel of Corti and reach the outer hair cells at the level of the inferior pole of the cells. The fibres then turn at right angles basalward, forming the external spiral bundle. These fibres have been thoroughly described by many authors; they are systematized as one bundle for each row of sensory cells. The fibres change their course constantly passing from one bundle to the next. The length of individual fibres is as much as one-third of a turn. Held gives the most accurate description of their manner of innervation. According to his observations, there are two kinds of fibres: branched (see 5, in Fig. 1) and unbranched (see 6, in Fig. 1). The latter innervates one sensory cell while the former innervates several cells. The branched innervation predominates by far over the unbranched. In other words, there is a finely localized and a largely diffuse innervation of the outer hair cells. On the basis of innervation, he described two kinds of outer hair cells, those innervated by a single fibre and those receiving multiple innervation. There is not a systematic distribution of these two types, but they are intermingled with each other.

The original purpose of the present work was to establish as accurately as possible the size, length and systematization of the radial and spiral fibres and to incorporate the new concept of innervation determined by the discovery of Rasmussen. Later, during the histopathological studies of cochleas of animals subjected to acoustic trauma, our interest turned also to the effect of this injury upon the efferent fibres or terminals of the tract of Rasmussen.

METHOD.

The studies were carried out in young guinea pigs (seven to 30 days old). After perfusion of the animal with formalin, the cochleas were carefully dissected from the temporal bones. The pyridine and silver nitrate method of Cajal or the silver impregnation method of Bodian was used. With the technique of Cajal, the specimens were stained in block. Following dehydration and clearing, the cochleas were dissected under a binocular dissecting microscope (20×). The bone of the apical turn was first removed until the organ of Corti and surrounding structures were freed from the bony capsule; then the apical turn was removed from the modiolus. With fine, sharp instruments, Reissner's membrane, spiral ligament, limbus and modiolus were taken away. When the cochlea was properly dissected, the complete organ of Corti with the basilar membrane retained its normal spiral shape. In order to avoid superimpositions, each spiral turn was divided into two or three pieces and mounted in balsam. The dissection was continued in the same way, turn after turn, until all of the cochlear duct was mounted on slides for study. When the method of Bodian was used, the specimens were fixed and stained in block; the cochleas were dissected as before and each piece was toned separately.

With training and with appropriate instruments, it is possible to dissect long pieces of the organ of Corti from the basilar membrane and surrounding structures. We used dissection because with this method the cochlea is represented as an uncoiled ribbon and it is easier to follow and measure the individual nerve fibres and to study their distribution at different levels. In serial sections of embedded material this is accomplished only with difficulty, because it is not always possible to trace the course of individual fibres.

The effects of degenerative processes of the cochlea on the intraganglionic spiral bundle were observed in stained and mounted sections of animals' cochleas used in studies on acoustic trauma. The animals had been briefly exposed at very high intensities and then kept alive for periods between three and four months. The material had been stained by the hematoxylin-eosin method. Although this method is not the proper one for the study of nerve fibres, it gives valuable information about the shape and number of cell bodies and fibres as a whole in the ganglion of Corti. The study showed that promising results could be expected by the use of silver stains in cochleas traumatized acoustically or subjected to other experimental methods.

RESULTS.

(A) The Radial Fibres.

In the preparations, it was possible to follow some of these fibres from the ganglion of Corti to the inner hair cells (see 1, in Fig. 2), where they innervate several neighboring sensory cells. This is in agreement with the classical descriptions. The innervation of the inner hair cells is so compact that, as Held pointed out, it is not possible with the method used to follow accurately all of the individual fibres. For this purpose, it is necessary to resort to other methods of staining. It is clear, however, that the plexus formed by the nerve endings around the inner hair cells is remarkably more pronounced than on the outer hair cells.

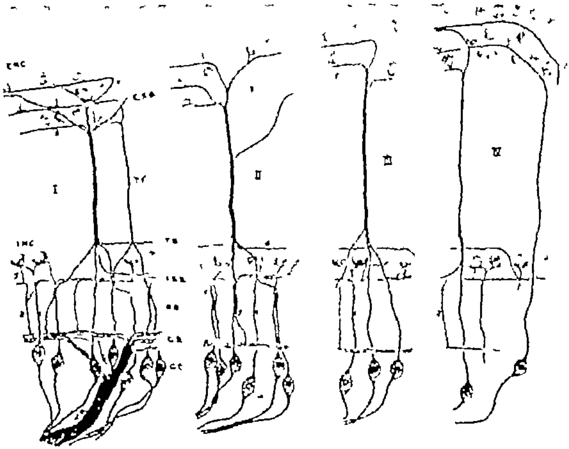

Fig 2 The diagram represents the innervation of the cochlea of the guinea pig at different turns. It is based on our own observations and on those of Lorente de Nó, Held and Rasmussen. I, first turn. II, second turn. III, third turn, IV, apical turn. This figure is substantially the same as Fig. 1, but it emphasizes the fewer fibres of the intraganglionic spiral bundle above the first turn, the internal spiral bundle and the formation of fasciculi in the tunnel. Abbreviations are the same as in Fig. 1.

(B) The Spiral Fibres.

1. The intraganglionic spiral bundle. The terminals of the tract of Rasmussen have a spiral course within the ganglion of Corti anatomically identified as the intraganglionic spiral bundle. The size and position of this bundle was determined in the adult guinea pig in both silver impregnated and hematoxylin-eosin stained material. The results were then compared with the animals subjected to acoustic trauma. The serial sections showed that the tract of Rasmussen enters the ganglion of Corti at about 3 mm. from the round window and breaks up into several fasciculi of different sizes and at different positions. After a short radial course some of them bend to the round window, others to the apex. Later the different fasciculi join together to form one bundle or a group of bundles. The bundle is found constantly in the outer part of the ganglion as is shown in Figs. 3, 5, 6, 7 and 8. The cross-sectional area of the bundle at 4 mm. from the round window (see Fig. 3) is 0.012 mm^2. At 7 mm. (see Fig. 5) it is about 0.005 mm^2. with borders clearly separated from the other structures. At the basal part of the second turn (about 12 mm. from the round window) the area is reduced to about 0.003 mm^2. or less (see Fig. 7). From this point on, the bundle becomes smaller and smaller, and it is very difficult to distinguish it from other structures in the ganglion; in the apical turn it is only visible in tangential sections. The reduction in the cross-sectional area is caused by the collaterals and terminals constantly being sent to the organ of Corti. The tract of Rasmussen is shown in Fig. 2 entering the cochlea in the first turn and breaking up into fasciculi to form the intraganglionic spiral bundle (G.B.). The figure also shows the greatly diminished size of the bundle as it reaches the apex.

In guinea pigs subjected to acoustic trauma and kept alive for long periods of time, atrophy of the organ of Corti and the primary neurons was found. The extent and location of damage varied with the frequency and intensity of the sound used, but the intraganglionic spiral bundle remained approximately normal in shape and size (see Figs. 4, 6 and 8). Measurements made in a few specimens showed a slight reduction in the sizes of the bundle in the basal part of the first turn. Unfortunately, there was no experimental material available which showed atrophy of the primary neurons to the end of the second, third, or apical turns.

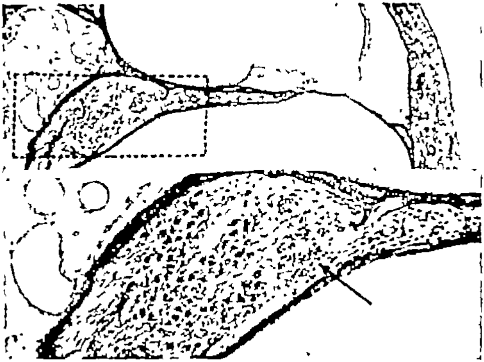

Fig 3. Upper: Photomicrograph of normal basal turn at 4 mm. from the round window. Hematoxylin-eosin. 48×.

Lower: Photomicrograph of the area of the ganglion of Corti enclosed in the rectangle of the upper figure. The arrow points to the intraganglionic spiral bundle formed by several groups of fibres with their borders clearly outlined. 130×.

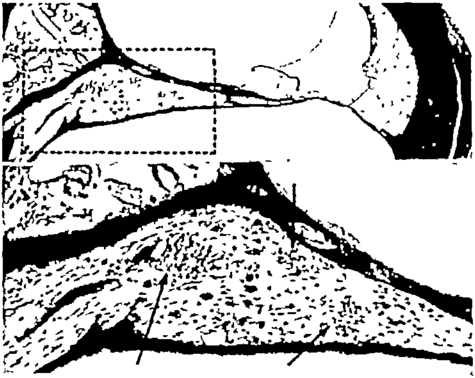

Fig 4 Upper Photomicrograph of pathological basal turn about 3 mm from the round window showing atrophy of the ganglion and organ of Corti. Hematoxylin-eosin 55×

Lower Photomicrograph of the area of the ganglion of Corti enclosed in the rectangle of the upper figure The cells of Schwann remain and also a few ganglion cells. The arrows point to several fasciculi, situated at different places, with normal density of fibres This illustrates the position of the fasciculi immediately after the tract of Rasmussen enters the ganglion. Later they join together to form a single bundle. 130×.

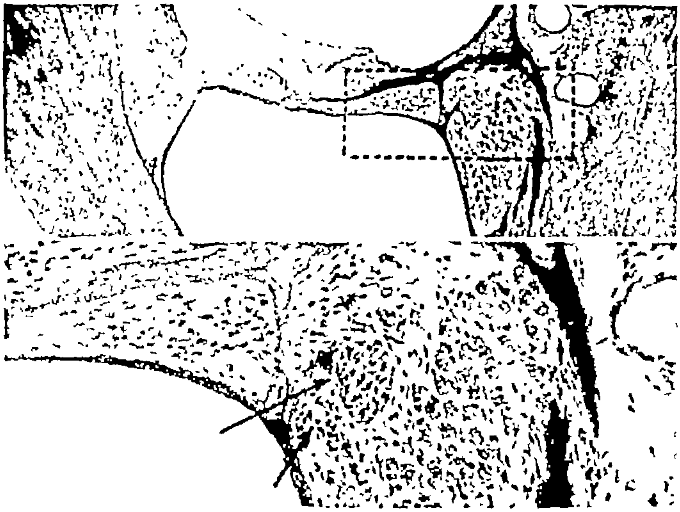

Fig. 5. Upper: Photomicrograph of the normal basal turn at about 7 mm. from the round window. Hematoxylin-eosin. 58×. Same animal as Fig. 3.

Lower: The arrow points to the intraganglionic spiral bundle formed by a big and compact fasciculus and two small ones. The total cross-sectional area of the bundle at this level is reduced to one-half of the size seen at 4 mm. from the round window. 180×.

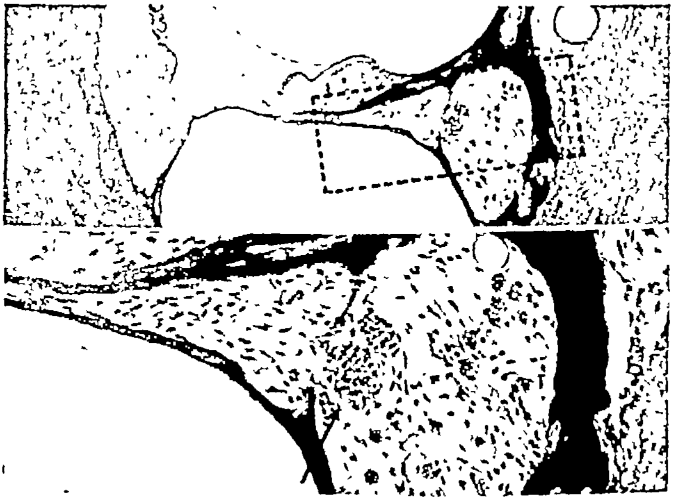

Fig. 6. Upper: Photomicrograph of basal turn, traumatized acoustically, at about 8 mm. from the round window. It shows atrophy of the ganglion and organ of Corti. Same animal as Fig. 4. Hematoxylin-eosin. 66×.

Lower: Higher magnification shows the cells of Schwann and a few ganglion cells. Arrow points to the intraganglionic spiral bundle formed by three fasciculi clearly outlined. The density of fibres is about the same as in the normal. 170×.

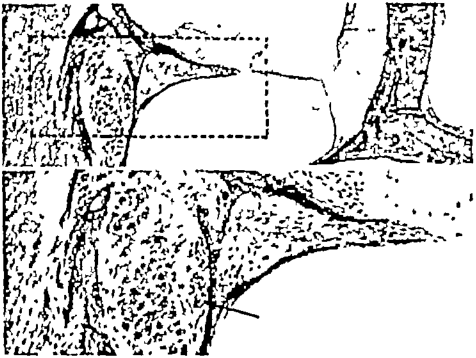

Fig 7 Upper Photomicrograph of normal ganglion and organ of Corti of the second turn at about 13 mm from the round window Same animal as Figs 3 and 5 Hematoxylin-eosin. 65×

Lower The arrow points to the intraganglionic spiral bundle which at this point is already reduced to one fourth of the size of the bundle at 4 mm. from the round window

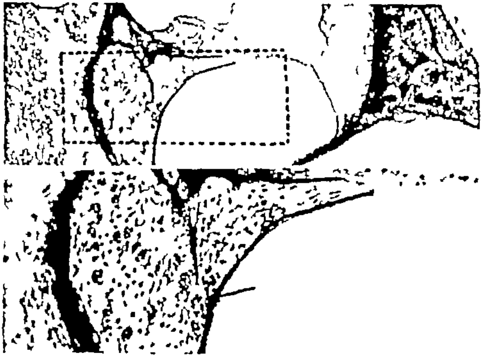

Fig 8 Upper Photomicrograph of pathological second turn at about 13 mm. from the round window Same animal as Figs 4 and 6 Teh organ of Corti is absent and the ganglion of Corti presents a pronounced degree of atrophy Hematoxylin eosin. 50×

Lower Schwann's cells remain and also a few ganglion cells The arrow points to the intraganglionic spiral bundle which is reduced to a few fasciculi of small size but with a total cross-sectional area of about the same size as the normal represented in Fig 7 lower 166×

The same pattern of atrophy of the organ of Corti and primary neurons was observed in slides in the collection of Schuknecht. His animals (cats) had received traumatic blows to the head. The intraganglionic spiral bundle remained approximately normal. Guild[8] described the persistence of the intraganglionic spiral bundle in humans after atrophy of the organ of Corti and primary neurons.

2. *The internal spiral bundle.* According to Lorente de Nó and others, this bundle runs apicalward underneath the inner hair cells. He also described it as terminals of the intraganglionic spiral bundle and stated that they innervate, almost exclusively, the inner hair cells. Our observations agree with these (see 3, in Fig. 2) although it was found that many fibres course basalward and many other fibres extend for distances of as much as 200μ or more in the internal spiral bundle. They then pass laterally to the outer hair cells. Lorente de Nó found the internal spiral bundle to be formed of very fine fibres. There is, however, a great variability in their diameters. Indeed, the finer ones (less than 0.5μ) are the most common, but there are also many with a diameter as great as the largest unmyelinated fibre (approximately 1.0μ) found in the cochlea.

3. *The tunnel spiral bundle.* This is formed by a small number of fibres coursing inside the tunnel of Corti. According to Held, they turn back to innervate the inner hair cells. It was found that the bundle runs in close relation to the inner pillars and is formed by a few fibres which are of small diameter (0.5μ). They extend either towards the round window or towards the apex (see 4, in Fig. 2). Some of them course for a distance of 300μ. All of them turn outwards to the outer hair cells. This fact confirms the possibility pointed out by Wever. In some specimens there was an increase in thickness of the bundle as the apex was approached. Fig. 11 illustrates the position and relative size of the bundle in relation to the internal spiral bundle in the apical turn.

4. *The external spiral bundle.* The present studies are in agreement with the observations of Held concerning the

course of these fibres and their innervation of the outer hair cells. There are two types of fibres, branched and unbranched, and also two kinds of cells, the singly and the multiply innervated. In only a few instances were unbranched fibres and singly innervated cells observed. The origin of the external spiral fibres is from different sources: *a.* directly from the habenula perforata; *b.* some fibres, after crossing the habenula perforata, bend either to the round window or apex for variable distances. These form in their course part of the internal spiral bundle before they turn outward to the outer hair cells; *c.* some collaterals, although exceptional, pass from the internal spiral bundle to the outer hair cells, and *d.* all fibres of the tunnel spiral bundle bend at right angles to the outer hair cells.

The course of the fibres in the tunnel is in two different positions; one crosses in fasciculi horizontally about the middle of the tunnel (medial fibres); the other crosses in single units or groups of two fibres at the bottom of the tunnel [basilar fibres (see Fig. 9)].

In the first and second turns, the fasciculi are formed by five to six fibres each (see Figs. 2, 9 and 10), but single units and fasciculi of two or three fibres are also intermingled with them (see Fig. 12). The fibres of the fasciculi come from different places as has been already described and as illustrated in Figs. 2 and 12. As soon as they cross the inner pillars, they usually join into a very tight and twisted fasciculus. When the fasciculus reaches the outer hair cells it breaks into single units which form the external spiral bundle. Most of the single units run basalward but many were also found to course in the opposite direction as illustrated in Fig. 13. The number of fasciculi which traverse the tunnel and the number of fibres in each fasciculus diminish as the apical turn is reached. In this turn, usually neither fasciculi nor single units are found to traverse the middle of the tunnel.

There is no information concerning the number of fibres to the outer hair cells at different levels. The present techniques do not stain all of the fibres.

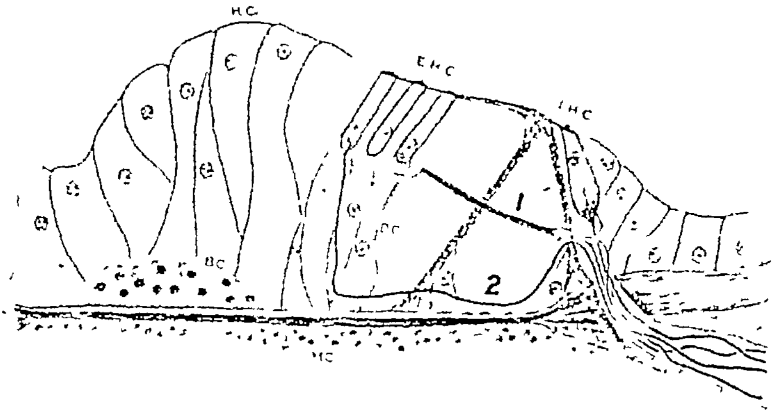

Fig. 9. Diagram of the organ of Corti in cross-section. It shows the structures and innervation as seen in the basal half of the first turn of the young guinea pig. I.H.C., inner hair cells; E.H.C., outer hair cells; H.C., Hensen's cells, D.C., Deiter's cells; B.C., Böettcher's cells; M.C., mesothelial cells. (1) Medial fibres crossing the tunnel in one fasciculus of four fibres, two coming from the habenula perforata, one from the internal spiral bundle (represented by dots underneath the inner hair cells), and the fourth one from the tunnel spiral bundle (represented by dots inside the tunnel close to the inner pillar). (2) Basilar fibre coming from the habenula perforata; in the tunnel it runs over the basilar membrane and then crosses to Deiter's cells, where it bends at right angles away from the basilar membrane toward the outer hair cells. The dots underneath the outer hair cells represent fibres of the external spiral bundle.

The basilar fibres (see 2, in Fig. 9) are fine fibres (less than 0.5μ) which pass between the inner pillars at the same level as the medial fibres. They extend across the tunnel immediately above the basilar membrane in single units or groups of two. They pass out of the tunnel at the foot of the outer pillars (see Fig. 14). Some course between Deiter's cells, bend at right angles and ascend to the outer hair cells, others bend when Deiter's cells are reached. They are present throughout the organ of Corti but their density increases as the apex is reached. At the apical turn almost all fibres to the outer hair cells are of the basilar type. After reaching the outer hair cells, the course of the fibres follows the same principle already described for the medial fibres. The exact information as to their endings or their origin behind the habenula perforata is not available. The particular arrangement of

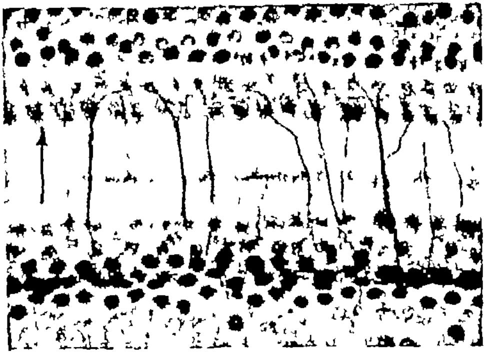

Fig 10 Photomicrograph illustrating the fasciculi in the tunnel of Corti in the second turn It shows several fasciculi of different density Because of the thickness of the specimen many fasciculi and single fibres are out of focus The arrow points to the outer hair cells. Bodian's method. 600×.

fibres in the tunnel of Corti in young guinea pigs was confirmed by Guild and for adult animals in the present studies. Toward the round window the medial fibres predominate while the basilar fibres are predominant toward the apex.

The central and peripheral myelinated processes of the ganglion cells have a diameter of 2.5μ to 3μ. Among the peripheral fibres there are many of small diameter which, in a few instances, were identified as collaterals of the intraganglionic spiral bundle. The central or peripheral processes were never found to exceed 3μ in diameter. The diameter of the unmyelinated fibres varied between less than 0.5μ and a maximum of 1.0μ. The smallest fibres were found in the internal spiral bundle, in the tunnel spiral bundle and among the basilar fibres crossing the tunnel to the outer hair cells. Those of greatest diameter were found in the internal spiral bundle and principally among the medial fibres which pass through the tunnel of Corti to the outer hair cells. There is no evidence of a systematic distribution of the fine and coarse

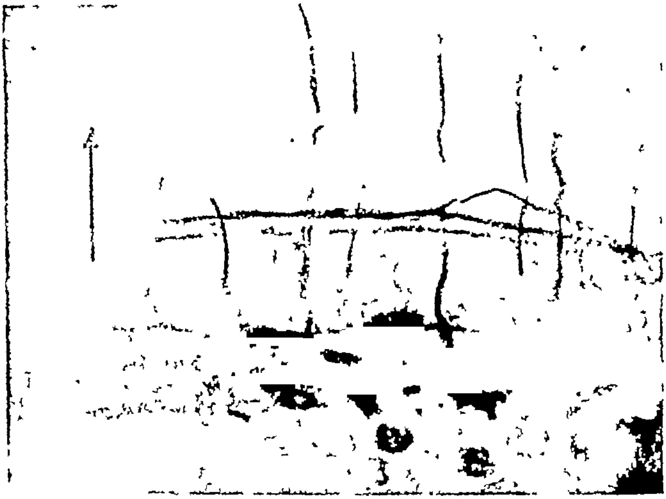

Fig 11 Photomicrograph illustrating the tunnel spiral bundle in the apical turn The arrow points to the outer hair cells. The bundle is formed by groups of few fibres crossing the tunnel on the upper part of the figure. To the left one fibre is turning out to the outer hair cells In the lower part of the figure is the internal spiral bundle. Notice the great difference in size between the two bundles The inner hair cells are out of focus Single units or groups of two fibres are crossing to the outer hair cells Cajal's pyridine method 1260×

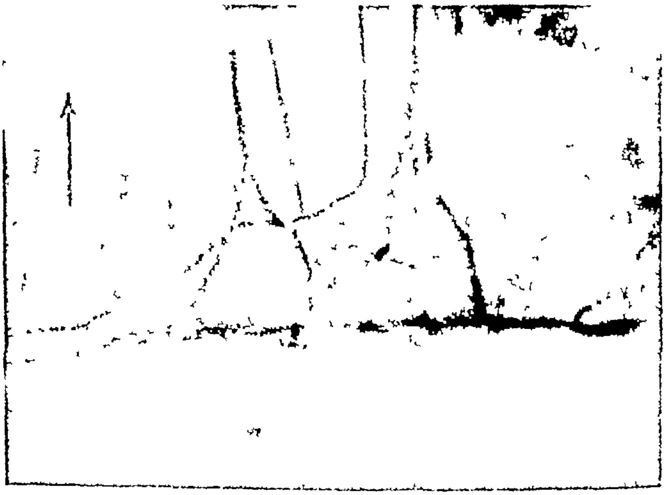

Fig 12 Photomicrograph the origin of the fasciculi in the tunnel of Corti in the first turn It shows two fasciculi, each formed by many fibres coming from different places, and also, intermingled with them, single units and fasciculi of two or three fibres In this area there are other fibres out of focus because of the thickness of the specimen The arrow points to the outer hair cells Bodian's method. 1400×.

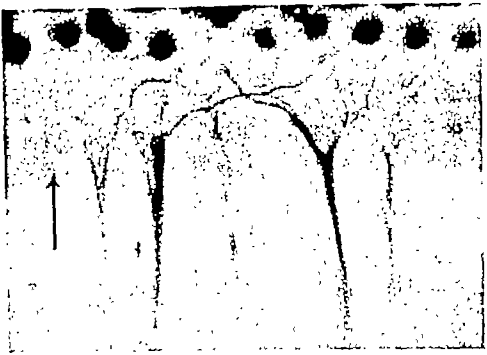

Fig. 13. Photomicrograph: the endings of the two fasciculi represented in Fig. 12 as they reach the outer hair cells. The fasciculi break up into individual fibres which take a spiral course toward either the apex or the round window. Bodian's method. 1260×.

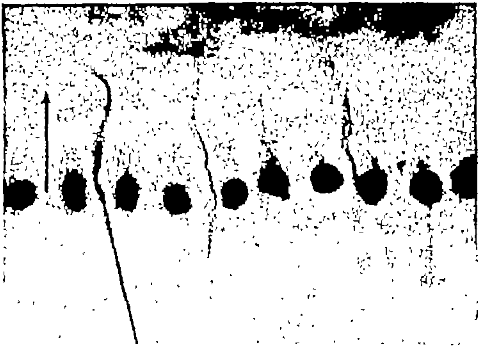

Fig. 14. Photomicrograph taken at the same place as Fig. 13 but focused at the foot of the outer pillars. It shows the nuclei of the outer pillar cells and single fibres or group of two fibres crossing between them to the outer hair cells. The arrow points to the outer hair cells. Bodian's method. 1260×.

fibres. In general, it may be said that toward the round window there is a predominance of coarse fibres while toward the apex the fine fibres are the most numerous.

The length of the fibres is extremely variable. The shortest are the radial fibres to the inner hair cells, whose myelinated processes measure about 1 mm. and the unmyelinated portions 30μ. The longest are the intraganglionic spiral fibres to the apex, whose unmyelinated parts were followed for about 300μ. The myelinated portions were calculated as about 10 mm. in length in Rosenthal's canal, and in addition, there is an initial portion of at least 10 mm. between the cochlea and the contralateral superior olivary complex. It has been impossible to establish with complete accuracy the lengths of the various fibres. The measurements are, however, in general agreement with the classical descriptions.

DISCUSSION.

In consideration of the discovery of Rasmussen the innervation of the cochlea should be regarded as formed by two different systems, namely, efferent and afferent.

The efferent system in the cochlea is the peripheral part of the tract of Rasmussen. The report of Rasmussen and the findings in animals with degenerative processes of the organ and ganglion of Corti have a triple interest:

a. Anatomically, the origin of the intraganglionic spiral bundle has been identified in the superior olivary complex of the opposite side. The cell bodies and dendritic processes are present in this nuclear mass and not in the ganglion of Corti as previously described. The possibility of some intraganglionic spiral fibres with cell bodies inside the ganglion is not denied. The localization of the nerve endings of the efferent fibres is a point to be clarified carefully because of the physiological implications. Lorente de Nó describes some fibres passing from the intraganglionic spiral bundle to the outer hair cells which the present studies confirm. The extent of this innervation on the outer hair cells is a question still to be answered.

Since the tract of Rasmussen enters the cochlea as a whole at the level of the first turn, and runs toward the apex, it is evident that some fibres in the intraganglionic spiral bundle are as long as the cochlea. The longest efferent fibre has the length of the tract of Rasmussen (to be determined in different animals) plus the intraganglionic course of the tract up to the apex (about 10 mm.), plus the spiral course underneath the inner hair cells (some of these were followed for 300μ). More important than the length is the ramifications of the efferent fibres. This is still to be determined.

b. Pathologically, it is of interest that atrophy of the primary neuron can occur in the presence of an almost normal intraganglionic spiral bundle. It is probably because the metabolic centers of the efferent fibres are far removed from the degenerative process. There is, however, a small reduction in the cross-sectional area of the bundle in the presence of atrophy of the primary neuron. It is not an artifact introduced by the plane of the section, because the difference, although small, is constant. This is a problem for further studies.

c. Physiologically, the fibres of the tract of Rasmussen presumably carry impulses from the superior olivary complex to the cochlea of the opposite side. The first suggestion of its function appears in the studies by Galambos *et al.*,[8] who found physiological evidence of a cochleocochlear interaction in the cat. As to whether this phenomenon is accomplished through the tract of Rasmussen, these authors state, "it is neither supported nor denied by the information at hand." The evidence for the efferent character of the tract of Rasmussen is at present almost entirely anatomical. The observations of Galambos *et al.* indicate rather strongly, however, that there are efferent fibres to the cochlea. In the absence of other known efferent fibres, this is significant circumstantial evidence supporting Rasmussen's anatomical interpretation.

It is remarkable that the size of the spiral bundle reduces suddenly at about the second turn. This fact suggests that whatever the function of these fibres may be, it is probably more effective in the first turn. Since the first turn in the guinea pig is about one-half of the total length of the cochlea, the distribution of the terminal fibres of the tract of Rasmussen may have an anatomical rather than a physiological basis.

The afferent system is formed by two types of fibres, the radial and the spiral fibres.

To the radial fibres belong those which innervate a single or a small group of sensory cells. They are the radial fibres to the inner hair cells described by von Ebner and Lorente de Nó and the unbranched fibres to the outer hair cells described by Held. The radial fibres to the inner hair cells show a constant and systematic distribution throughout the cochlea while the radial fibres to the outer hair cells (unbranched fibres of Held) do not show a similar arrangement. Furthermore, the density of the unbranched fibres at different levels still is unknown. In any event, whatever the function of the radial fibres is, a principle of discrete localization exists for both types of sensory cells although it is more pronounced for the inner hair cells.

To the other type of afferent fibres, the spiral fibres, belongs the external spiral bundle with extensive ramifications to the outer hair cells. From the classical descriptions and the present observations, this is the predominant type of innervation on the outer hair cells. The functional significance of the diffuse innervation by the afferent spiral fibres is not known. Davis[10] suggests that it may be to allow spatial summation of weak local excitatory effects when the cochlea is stimulated by very faint sounds. It is doubtful that the inner hair cells have an afferent spiral innervation, since the internal spiral bundle, whose fibres give off numerous collaterals, is formed only by efferent fibres; therefore, the only afferent innervation of the inner hair cells is by the radial fibres. In other words the outer hair cells have principally a diffuse and overlapping innervation and the inner hair cells a more discrete or localized innervation. The inner hair cells are innervated by both the efferent and afferent systems while the outer hair cells are innervated almost exclusively by the afferent system.

SUMMARY.

The innervation of both the internal and the external hair cells is complex. In each case there are two classes of nerve fibres, the "radial" and the "spiral."

The radial fibres run directly outward from the spiral lamina and each fibre innervates one, two or, at most, three hair cells. The radial fibres form the chief nerve supply of the inner hair cells but they are in a minority in the supply of the external hair cells. Their cell bodies lie in the spiral ganglion (Corti).

The spiral fibres run for distances up to several millimeters in both directions along the organ of Corti. Each fibre innervates many sensory cells, although a given fibre does not innervate every hair cell along its course. A branch to every second or third cell is more typical. This type of innervation is predominant for the external hair cells. These spiral fibres form the well-known "external spiral bundles."

A given external hair cell may be innervated by more than one spiral fibre. A given inner hair cell may receive endings from sometimes one, usually two or, at most, three radial fibres. In addition some, at least, of the inner hair cells also receive endings from spiral fibres, and external hair cells receive some radial fibres in addition to their spiral fibres.

The spiral fibres to the external hair cells are afferent, with cell bodies in the spiral ganglion. The spiral fibres to the internal hair cells, however, seem to be *efferent* fibres. Their cell bodies lie in the medulla oblongata in the superior olivary complex of the *opposite* side. They run from this nucleus in the tract of Rasmussen and form a compact bundle in the auditory nerve. This bundle is clearly marked in the basal turn where it forms the intraganglionic spiral bundle. The collaterals terminate on internal hair cells. This bundle and the fibres of the tract of Rasmussen do not degenerate when the organ of Corti is destroyed by acoustic trauma.

The double innervation of the hair cells can be summarized as follows:

		TYPE OF INNERVATION	
		Dominant	Secondary
HAIR CELLS	Inner	RADIAL	SPIRAL
		Afferent	*Efferent*
	Outer	SPIRAL	RADIAL
		Afferent	Afferent

I express my appreciation to Dr. Walter P. Covell, Department of Otolaryngology, Washington University, for his technical advice and for furnishing the serial sections of animals traumatized acoustically; to Dr. Harold Schuknecht of the University of Chicago for furnishing me with his histopathological collection and for his criticisms; and to Dr. Stacy Guild for giving me the opportunity to examine his remarkable collection, for his criticisms and for his suggestions.

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PRIMARY CARCINOMA OF EXTERNAL AUDITORY CANAL, MIDDLE EAR AND MASTOID.*†

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INTRODUCTION.

Until recently, primary carcinoma of the external auditory canal, middle ear and mastoid has been considered as a medical curiosity. In reviewing the literature, I have found 211 authentic cases. It is quite possible that many more have been seen but have not been reported. Involvement of the auricle and external auditory canal are more apt to be seen by the dermatologist and radiologist but seldom by the otologist. According to statistics, the average practicing otolaryngologist will see one or, at the utmost, two cases of this distressing disease during a lifetime. I am fortunate in having seen my allotted number rather early in my career.

INCIDENCE.

The types of malignant tumors of this region run the gamut of new growths, ranging from hemangioendotheliomas to squamus cell and adenocarcinomas. The most rare are the adenocarcinomas, and Furstenberg,[6] Lukens[12] and Grabscheid[7] have reported cases of these. The squamus cell carcinoma is the one most frequently found. According to various authors, the frequency of occurrence varies from one in 5,000 to one in 20,000 of otologic cases. The condition is rare in persons under 40 years of age. It is equally distributed among the sexes. Grossman *et al.*[8] reported six cases, all in white females. The disease is exceptionally infrequent in the Negro race.

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ETIOLOGY.

The etiology of carcinoma of the ear is still obscure; however, certain associated and predisposing factors have been suggested as precursors to carcinoma. These are: *1.* chronic otorrhea, either from otitis media or mastoiditis; *2.* chronic eczema and other chronic dermatoses; *3.* senile keratoses; *4.* cutaneous horns; and *5.* chronic ulcerations from trauma or frostbite.

Although there is a wide diversity of opinion as to the relative significance of the various etiologic factors, most authors seem to stress the presence of a chronic aural discharge as a factor of great importance in carcinoma of the external canal and middle ear. Guttman,[9] on the other hand, asks "why we blame irritation of chronic discharge when there are so many chronic discharging ears that never become malignant." Figi and Hempstead[5] reported two cases of carcinoma following repeated attacks of erysipelas with ulceration of the external ear and canal. Danziger[1] reported cases in which "the condition was not preceded by chronic suppuration but occurred subsequent to acute otitis."

SYMPTOMATOLOGY.

The symptoms of carcinoma of the external auditory canal, middle ear and mastoid vary with the type, duration, size, location and invasiveness of the lesion. Basal cell carcinomas and adenocarcinomas of the mixed tumor type have aural symptoms of longest duration. Squamous cell carcinomas, especially of the middle ear, have the shortest duration of symptoms. The following are the most constant and frequent symptoms and findings that present themselves in this disease:

1. Chronic Aural Discharge: This is usually of many years' duration and is considered by Newhart,[15] Furstenberg[6] and Schall[21] to be of utmost significance when this discharge changes from a purulent to a bloody form. In the series of Figi and Hempstead[5] only 11 out of 38 cases had an aural discharge.

2. Pain: Pain in or about the ear is a very frequent complaint. It is severe and out of all proportion to the clinical findings. It usually follows the distribution of the trigeminal nerve and is sometimes aggravated by the opening and closing of the mouth. Furstenberg,[8] Schall[21] and Grossman *et al.*[9] believe that the pain is due to pressure of the growing tumor. Barnes[1] attributes the pain to infection of the tumor mass and skin lining the external auditory canal. Others favor the early invasion of the perichondrium and periosteum as the cause of this symptom. If the carcinoma arises in the middle ear or mastoid, the pain will present itself relatively early, but if it has its origin in the external auditory canal and invades the middle ear and mastoid secondarily, pain will be a very late symptom. According to Shuster,[22] the presence of intense pain suggests a malignancy, since a chronically discharging ear does not usually have pain.

3. Aural Growths: Occasionally a patient may seek medical advice because of this symptom alone. It is often very difficult clinically to differentiate between aural polyps, granulations and a malignancy. Schall,[21] Lederer[11] and other authors stress the suggestiveness of malignancy in any polyp or granulation tissue that bleeds profusely when removed or manipulated.

4. Ulceration: Ulcerations of the external auditory canal which persist and become worse in spite of treatment are very suggestive of an underlying neoplasm.

5. Palsies of the Various Cranial Nerves: Buckman[3] states that facial paralysis occurs frequently enough to be considered a symptom of carcinoma. When this is seen, the lesion is usually far advanced. Occasionally the IXth, Xth, XIth and XIIth nerves may become involved by the neoplasm, giving a jugular foramen syndrome with dysphagia, inarticulate speech and hoarseness. With invasion of the petrous pyramid there may at times be a paralysis of the IIIrd and VIth nerves.

6. Deafness and vertigo are infrequent symptoms. The deafness is usually proportionate to the amount of obstruction of the canal and involvement of the ossicular chain.

DIAGNOSIS.

"Successful treatment of carcinoma, regardless of its location, depends on early diagnosis. This is especially true in the middle ear, for by the time the lesion is grossly visible, invasion of the underlying bone has often already occurred" (House[10]). A diagnosis of suspected malignancy can be made from a careful history, the symptoms and significant clinical findings. A biopsy, however, is essential and, if negative, should be repeated as often as feasible. House[10] suggests the use of the Papanicolaou technique in all chronically discharging ears and also where a biopsy is not possible.

X-ray examination of the temporal bone is of little value in the early diagnosis of carcinoma; however, it is of inestimable value in determining the extension and bony involvement by the disease — a factor which is very important if surgery is contemplated.

PATHOLOGY.

Several authors have attempted to classify carcinoma of the ear into extrinsic and intrinsic forms. Lederer[11] has suggested the tympanic membrane as a natural anatomical delineation for clarifying these lesions of the ear. Schall[21] states that "a neoplasm in the external auditory canal beyond the isthmus is a potential malignant growth of the middle ear."

Metastasis of carcinoma of the ear to distant body organs is rare. Regional lymph nodes are often enlarged, but sections of them usually reveal a chronic inflammatory reaction. The disease is dangerous mainly because of its position and tendency to invade adjacent vital structures. The dura and the labyrinthine capsule are uniquely resistant to invasion by the neoplasm until very late in the course of the disease. House,[10] Towson and Shofstall[24] have reported cases of intracranial extension. Erosion of the internal carotid with fatal hemorrhage has been observed. Cranial nerve involvement other than the facial has been seen by several authors. Extension to the temporomandibular joint and parotid gland occurs relatively early.

A number of authors stress the fact that the lining membrane of the middle ear will undergo a change, with a change in the character of the suppuration. Panse[14] states "that a neoplasm which originates in a closed tympanic cavity probably results from a metaplasia of the cylindric cells of the lining membrane to the squamous cell type or has its origin from the glandular elements within the cavity."

TREATMENT.

The consensus of the opinion among all the authors is to treat this distressing disease with radical surgery, where feasible, and irradiation. Figi and Hempstead[8] report good results in their series, using electrosurgery and intensive irradiation by radium points directly to the site of the lesion. Others have used radical surgery and external Roentgen radiation. Schall,[21] in 1935, pointed out that "in the external use of irradiation, the clinical fact is ignored that with a dosage sufficient to cause necrosis of the bone, it may have no effect on the tumor invading the bone." He also suggested the following procedures:

1. Excision of the entire cutaneous canal.

2. Radical mastoidectomy for the purpose of inspection and for sufficient space for proper application of radium.

3. Irradiation must be applied at the site of the lesion.

Post-treatment follow-ups should be done at frequent intervals. Papanicolaou smears may indicate the progress of the disease.

PROGNOSIS.

According to Figi and Hempstead,[8] the prognosis of carcinoma of the external auditory canal, middle ear and mastoid is comparable to malignancy of the paranasal sinuses. In Schall's[21] series of five cases there was no recurrence in two to four years, but at the end of seven years all were dead except one, a hemangioma. In the series of 38 cases of Figi and Hempstead,[5] 20 patients lived two years, 15 lived three years, 10 lived seven years, eight lived eight years, three lived 10 years, and one lived 15 years.

CASE REPORTS.

I wish to present briefly five previously unreported cases of *squamous cell carcinoma of the external auditory canal, middle ear and mastoid* from the files of the University Hospitals. I have had personal contact with Cases 1 and 2 only.

Case 1: Mrs. M. D., a white female, age 56, came to the O. P. D. on Aug. 25, 1950, because of itching (five years' duration) and progressive deafness (two years' duration) of the right ear. There was no aural discharge at any time. Examination revealed a severe desquamating external otitis of the right ear with a pale red mass occluding the lumen of approximately the inner half of the external auditory canal. This mass bled easily when manipulated. There was tenderness in the right temporomandibular joint on palpation and pain on opening and closing of the mouth. Biopsy was deferred until the external otitis was under control. On Sept. 14, 1950, a biopsy of the mass was taken and was reported as a well differentiated squamous cell carcinoma.

The patient was admitted into the hospital on Sept. 16, 1950. Physical examination again revealed the above findings and also a bilateral mixed type deafness, worse on the right ear, a ventral hernia, and a mild hypertension. Laboratory findings were within normal limits. The serology was negative.

The X-ray report on the day of admission was "essentially negative examination of the mastoid regions with slight diminution and radiolucency of the right mastoid cells and no evidence of bone destruction seen" (see Fig. 1).

On Sept. 20, 1950, an endaural radical mastoidectomy was performed on the right ear. The external auditory canal was excised, completely removing thereby the major portion of the tumor. A small amount of soft friable tissue was found in the hypotympanum, and there seemed to be some invasion of the tympanic plate and floor of the external auditory canal. The remainder of the middle ear, antrum and mastoid were relatively free of tumor. Preauricular nodes were not removed. The cavity was not skin grafted.

The pathological report of the tissues removed at surgery was well differentiated squamous cell carcinoma clinically of the external auditory canal and in bone (see Figs. 2A and 2B).

On Oct. 6, 1950, the patient was started on external Roentgen therapy. She received a total of 3,600 r. through one field. A herniarrhaphy was done on Nov. 1, 1950, and she was discharged on Nov. 9, 1950.

She was seen in the O. P. D. twice a week. Papanicolaou smears were taken every two weeks. So far all have returned as negative for malignant cells. The cavity is epithelializing nicely at the present time.

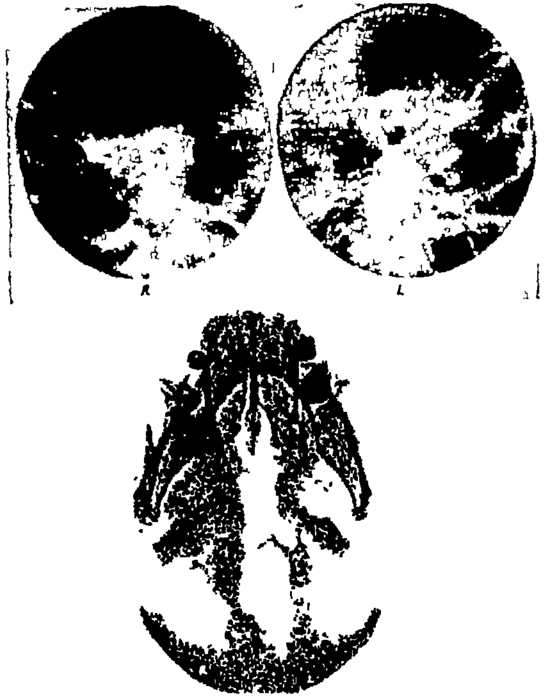

Fig 1 Case 1 Roentgenograms of both mastoids and petrous pyramids showing no bone invasion. Arrows indicate approximate areas where bone invasion was found during operation.

Case 2 Mr O K., a white male, age 72, was admitted into the hospital on Nov 8, 1950, with a complaint of chronic discharge from the right ear (35 years' duration) and severe pain in the right side of the face (one month's duration) The pain radiated to the temporal region zygoma and mandible It was aggravated by opening and closing of the mouth He had an "abscess" of the right ear on Oct. 24, 1950, with a bloody discharge Physical examination revealed the right external auditory canal almost completely filled with a pink granular mass The drum was not visualized The left ear, except for a very narrow canal and a small

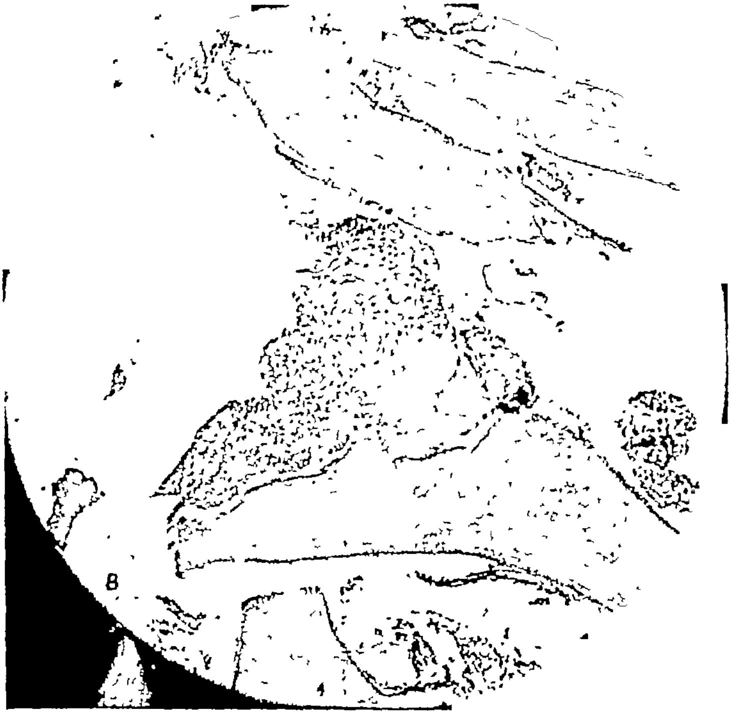

Fig. 2. Case 1 Biopsy of right external auditory canal showing well differentiated squamous cell carcinoma.

central perforation, was normal. There were no palpable regional nodes. He also had a moderate right facial weakness, a deformity of the right leg and foot, a mild hypertension and a left inguinal hernia.

A biopsy of the mass in the right external auditory canal was done on Nov. 9, 1950, and revealed a well differentiated squamous cell carcinoma (see Fig. 4).

Roentgen examination of both mastoids and petrous pyramids on Nov. 9, 1950, was reported as "there appears to be invasion and possibly erosion of the tympanic portion of the right ear by a carcinoma which is known to be present clinically. There is also mottling of the ear extending towards the tip of the petrous pyramid, probably due to invasion here as well" (see Fig. 3).

The laboratory findings were within normal limits. Due to the patient's age and X-ray evidence of extensive invasion, it was deemed inadvisable to perform any surgery. He was immediately started on external Roentgen therapy. He received a total of 3,500 r. through one field.

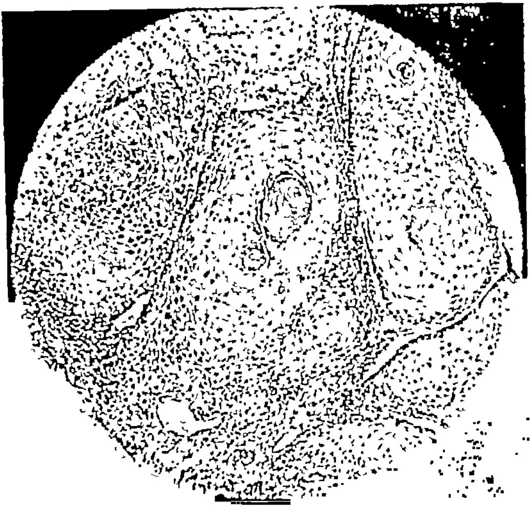

Fig. 3 Case 1A. Curetted biopsy from the right tympanic plate showing the carcinoma invading the bone.

A follow-up on Jan. 10, 1951, showed the canal dry and smooth, but markedly narrowed towards the drum. The drum was not visualized. The patient stated that he had some relief from the intense pain after the irradiation.

Case 3: Mr. J. B., a white male, age 31, was admitted to the hospital on March 31, 1939, with a complaint of a discharging left ear since the age of two years, severe pain in that ear and a growth in the canal of the left ear of five months' duration. Prior to admission he had four attempts at removal of the mass for the relief of pain. Two weeks prior to admission a fifth biopsy was done and revealed the mass to be carcinoma.

Physical examination showed a purulent discharge in the left external auditory canal and a red mass at about the junction of the bony and cartilaginous portions of the canal. The drum was not visualized. There was no hearing by air, and bone conduction was poor. The right ear was normal.

On April 1, 1939, the Roentgenographic report was a "malignant tumor originating in the left external auditory canal with considerable amount

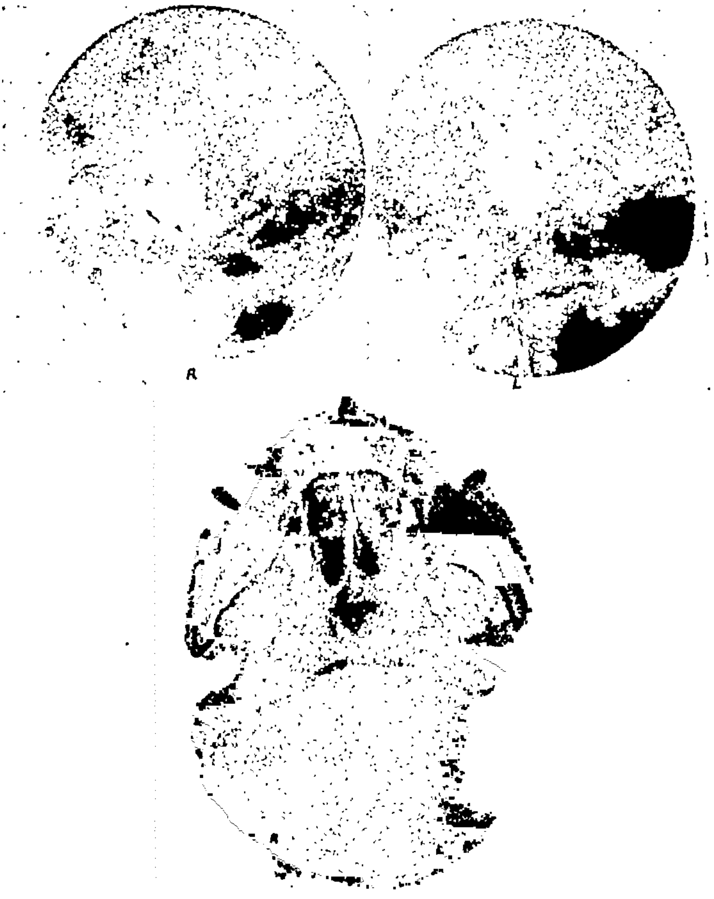

Fig. 3. Case 2. Roentgenograms of both mastoids and petrous pyramids showing invasion and erosion of the tympanic portion of the right ear and also a mottling of the bone extending towards the tip of the petrous pryramid.

of bony destruction around it. In left mastoid, cells are absent. In the region of the auditory canal there is an extensive area of bone destruction which measures 2 cm. by 1.5 cm. The area of bone destruction in the petrous bone is secondary to the tumor" (see Fig. 5).

A postauricular radical mastoidectomy with excision of the membranous external auditory canal was done on April 1, 1939. The major portion of the tumor was removed with the membranous canal. The middle ear was free of tumor tissue. "It was felt at the time of operation that the tumor tissues extended down to the middle ear, possibly invading the left temporomandibular joint."

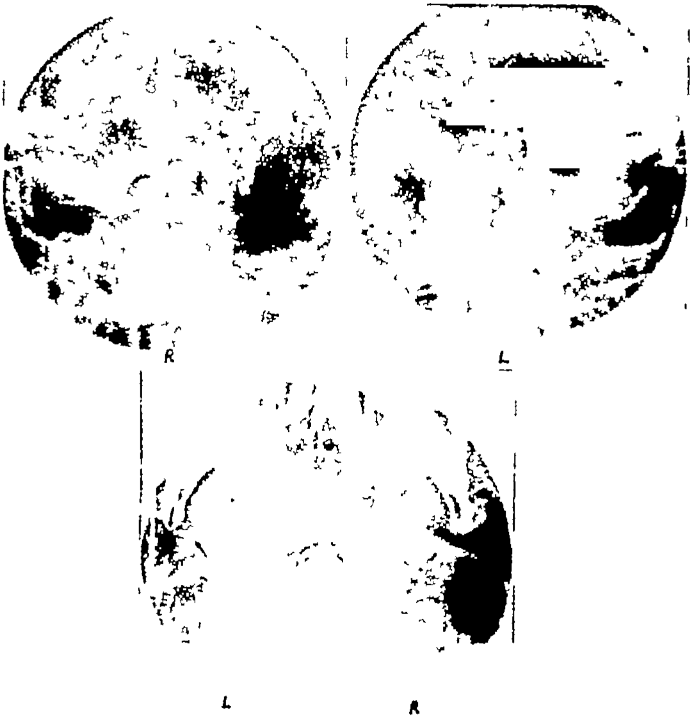

Fig 5 Case 3. Roentgenograms of both mastoids and petrous pyramids *showing extensive bony destruction in the left external auditory canal, mastoid and petrous pyramid.*

symptoms until about five months prior to admission. At that time she began to have pain, occasional bleeding from the canal, and difficulty in chewing because of pain in the right temporomandibular joint. She also complained of swelling behind and anterior to the right ear. A local doctor made sections of the "wart" on five occasions with no pathological report. Another doctor took a biopsy and reported it as "radioresistant carcinoma." The month prior to admission the patient had lost weight and had experienced severe and constant vertigo except when absolutely quiet.

Physical examination revealed a horizontal nystagmus on right lateral gaze. The right ear was tender, the mastoid area was swollen, the auricle protruded and was tender when pulled, and there was a large movable node anterior to the ear. There was a large, soft, pink smooth mass filling the right external canal. The tissue bled easily, and there was considerable purulent exudate in the canal. The left ear was normal

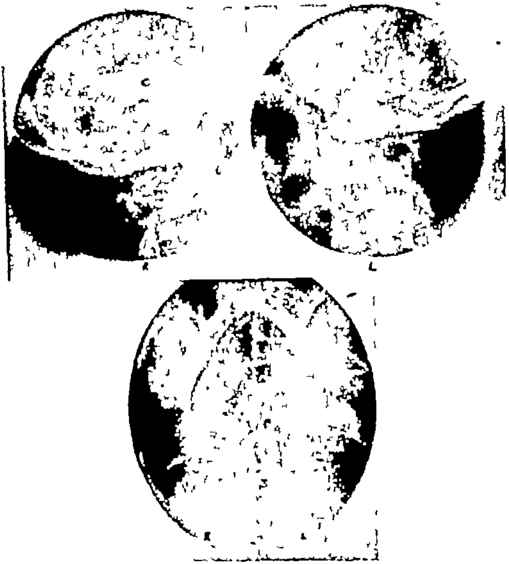

Fig. 6. Case 4. Roentgenograms of both mastoids and petrous pyramids showing extensive bone destruction involving the mastoid process of the right temporal bone, extending forward to uncover the right temporomandibular articulation and medially to destroy the lateral portion of the right petrous ridge.

There was no demonstrable hearing in the right ear. The rest of the physical examination was essentially normal except for varicose veins and discolorations of both legs.

The X-ray examination of the mastoids and petrous pyramids on Sept. 17, 1948, was reported as "extensive bone destruction involving the mastoid process of the right temporal bone, extending forward to uncover the right temporomandibular articulation and medially to destroy the lateral portion of the right petrous ridge" (see Fig. 6).

On Sept. 20, 1948, the major portion of the tumor mass and the right external auditory meatus were excised as a biopsy. The pathological report was "partially differentiated squamous cell carcinoma clinically of the external auditory canal."

A right preauricular abscess was incised and drained on Sept. 24, 1948, and she received a total of 4,437 r. through two fields.

After discharge from the hospital the patient was not seen again in either the O. P. D. or in any other department. The outcome of this case is, therefore, unknown.

Case 5: Mr. J. M., a white male, age 39, was admitted into the hospital on Aug. 11, 1940, with a complaint of severe and constant pain in the left ear of nine weeks' duration. At the onset of his symptoms he was seen by a physician who told him that he had a pimple in his ear and treatment was given. The pain was not relieved. He was again seen by his physician and at this time some bone was found to be exposed in the left external auditory canal. The patient received treatment for this condition, including some X-ray therapy, but without relief of the pain. The patient then had three molars extracted, a tonsillectomy and treatment for a sinus infection. He was finally referred to a local physician who took a biopsy of the lesion, which was diagnosed as a well differentiated squamous cell carcinoma.

The physical examination on the day of admission was essentially normal except for the left ear, which showed a very tender, red, swollen external auditory canal. There was a small area of bone visualized in the anterior canal wall and also along the floor of the canal. The entire membranous portion of the canal up to the drum seemed to be involved. Laboratory findings were within normal limits. Serology was negative. X-rays showed catarrhal mastoiditis on the left. On the day following admission the entire membranous portion of the external auditory canal was excised through a postauricular incision. The drum was left intact. Electric cautery was then applied to the remaining tissue of the canal.

The patient subsequently received a series of X-ray treatments at St. John's Hospital.

On Aug. 25, 1940, he was again admitted to this hospital because of a recurrence of his symptoms and the carcinoma. At this time a small, firm mass was noted just below the lobule of the left ear. This mass was adherent to the overlying skin. It was the opinion of the patient's physician that this was a metastatic lesion and that radical surgery at this time was inadvisable and the patient was discharged.

The patient subsequently died.

CONCLUSION.

The literature concerning carcinoma of the external auditory canal, middle ear and mastoid has been briefly reviewed.

Five new cases of squamous cell carcinoma of this region are presented.

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THE TONSILLECTOMY-POLIOMYELITIS PROBLEM. A REVIEW OF THE LITERATURE.*†

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Some progress has been made toward a better understanding of the tonsillectomy-poliomyelitis problem but sharp basic differences of opinion still exist despite the vast medical literature published on this subject.

Sheppard, in 1910, was the first to study the relationship of poliomyelitis and tonsillectomy.

In 1928, Ayer reported nine cases of bulbar poliomyelitis following tonsillectomy by a five to 10-day interval.

Aycock and Luther,[1] in 1929, reported a controlled study of 714 cases of poliomyelitis which revealed an excessive number of bulbar cases following tonsillectomy after a specific time interval. Of 217 cases (30 per cent) who had prior tonsillectomy, 16 had been operated seven to 18 days preceding the onset of the disease and all of these 16 cases were of the bulbar type.

Eley and Flake,[2] in 1938, studied 418 consecutive cases admitted to infants' and children's hospitals of Boston. Of the 287 spinal cases, eight (2.7 per cent) had been tonsillectomized seven to 30 days previously. Of the 131 bulbar cases, 17 (13 per cent) had tonsillectomy between the tenth and twentieth days preceding onset of the illness.

Stillerman,[3] in 1938, analyzed 686 cases of poliomyelitis admitted to Willard Parker Hospital during 1935 and reported

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*In this paper, tonsillectomy refers to either tonsillectomy, adenoidectomy or both.

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13 cases of poliomyelitis, nine of which were bulboencephalitic, developing 10 to 22 days after adenotonsillectomy.

The report of Krill and Toomey,[4] in 1941 on the K family, of Akron, Ohio, was followed by widespread publicity in both lay and medical publications. In the K family tonsillectomy was performed August 22 on five apparently healthy children. In less than three weeks all had developed bulbar poliomyelitis and three had died. The sixth and youngest child was not operated and remain well, though the virus of poliomyelitis was recovered from his stool. Only two scattered cases of polio were reported in Akron prior to the K family cases.

Francis, Krill, Toomey and Mack[5] studied these K family cases and concluded that the operative procedure precipitated the severe bulbar form of poliomyelitis in children who otherwise probably would have escaped with inapparent infections.

Cunning,[6] in 1949, concluded: "After 10 years' study of this subject, following over 17,000 poliomyelitis cases and 35,000 tonsillectomy cases, I am still of the same opinion that I was one year ago; namely, that no definite causal relationship between tonsillectomy and poliomyelitis has been established; therefore, I do not believe that tonsillectomy should be postponed indefinitely simply because the summer months are the months during which poliomyelitis is prevalent. If, however, there is a distinct rise in the poliomyelitis rate, bordering on epidemic proportions, in any community, all elective operations should be postponed.

"Until evidence of a conclusive nature is forthcoming, the surgeon should decide whether or not to perform tonsillectomies during the summer months and whether the dangers of delaying the operation are greater than the still undetermined risk of increasing susceptibility to poliomyelitis."

Faber[7] re-examined Cunning's 1946 and 1947 reports and found the incidence of bulbar cases more than twice as great in patients tonsillectomized within two months of onset as in nontonsillectomized patients.

Fischer, Stillerman and Marks,[8] in reporting the 1937 Toronto epidemic, concluded that among children between the ages of three and 12 years, acute poliomyelitis developed more often in those recently tonsillectomized than in others and that the increased incidence was due entirely to the excess of bulbar cases. *Among the children with poliomyelitis, they found the incidence of the bulbar form more than twice as great in those whose tonsils had been removed at any time previously as in those with tonsils intact.*

Toomey and Krill,[9] in reporting cases over a 14-year period at Children's Hospital, Akron, and one year (1941) at City Hospital, Cleveland, stated there were more individuals who had had tonsillectomies and adenoidectomies within 30 days prior to the onset of poliomyelitis who developed a bulbar type of the disease than could be explained on the basis of mere chance or random sampling.

Lucchesi,[10] in 1944, analyzed 432 cases of acute anterior poliomyelitis and found the incidence of bulbar and bulbospinal poliomyelitis was *significantly higher in patients without than in patients with tonsils and adenoids.*

John Anderson[11] studied the 1943 Utah epidemic and reported that 43 per cent of the bulbar and bulbospinal cases were preceded by a tonsillectomy within 30 days of the onset. The incidence of poliomyelitis was 2.6 times greater and the incidence of the bulbar and bulbospinal type 16 times greater in recently tonsillectomized children than in the general child population.

Campbell,[12] in analyzing 303 patients admitted to the Colorado General Hospital during the 1946 epidemic, found that previous tonsillectomy seemed to increase the individual's susceptibility, particularly children, to the bulbar type if poliomyelitis was contracted.

Roberts,[13] in 1946, summarized his views on 274 cases of poliomyelitis following recent tonsillectomy during the last 35 years, stating that the annual incidence of poliomyelitis in everyday life is 31 times greater than in the recently tonsillectomized population even during the poliomyelitis season.

Pederson,[14] in 1947, studied 492 cases of poliomyelitis in San Francisco (1941-1945) and reported 11 cases following tonsillectomy. He found the incidence of poliomyelitis following recent tonsillectomy was not greatly out of proportion to the ratio of disease to the general population during an epidemic year, but that when poliomyelitis did occur following tonsillectomy it was more apt to be bulbar in type. He also concluded that there was a two to one higher incidence of bulbar poliomyelitis in tonsillectomized than in nontonsillectomized patients.

Winborn and Stansbury[15] made a survey of 134 poliomyelitis cases admitted to Parkland Hospital during the first eight months of 1946. Their conclusions were that the incidence of poliomyelitis in tonsillectomized individuals is no greater than in those individuals whose tonsils have not been removed and the danger of contracting poliomyelitis following recent tonsillectomy is minimal.

Eschenbrenner,[16] in 1948, reviewed the literature, stating that the incidence of poliomyelitis probably is not appreciably greater following tonsillectomy, but certainly when a tonsillectomy patient develops poliomyelitis soon after operation, it is usually bulbar in type.

Kinney[17] surveyed 237 cases of poliomyelitis occurring in Cuyahoga County, Ohio, in 1946 and 1947. He stated that the relationship between tonsillectomy and the acquiring of polio is no more than casual and the public hysteria concerning this problem is unwarranted.

Wesselhoeft[18] formerly favored a ban on tonsillectomies during the summer months but has revised his opinion and now recommends that the operation be performed immediately after the close of school in June in Massachusetts.

In a personal communication, Dr. Wesselhoeft[19] states that in Massachusetts the State Board of Health furnished all hospitals with a bulletin every week during the summer giving the number and location of cases and makes a statement as to whether or not it is advisable to refrain from tonsillectomies.

Later in a city where it had been considered wise to refrain from tonsillectomies, the hospitals are advised to resume operations when the case rate has fallen in that locality.

Dr. Feemster,[20] Director of the Division of Communicable Diseases for the Massachusetts Department of Public Health, in a personal communication described the origin and method of operation of the above noted Massachusetts procedure which has given the hospitals a uniform policy, helped reduce confusion of the public and rendered the position of the otolaryngologist more comfortable.

Gaylord Anderson,[21] in 1950, studied the 1946 outbreak of poliomyelitis in Minnesota and concluded that the risk of developing poliomyelitis was at least three times as great among those undergoing tonsillectomy as among a comparable group not undergoing this operation and the risk of bulbar infection was 11 times as great.

Treynor,[22] writing in 1950 of the 1948-1949 epidemic in southwestern Iowa, could demonstrate no connection between tonsillectomy and poliomyelitis, spinal or bulbar, in the 374 hospital cases studied.

Miller,[23] at the American Medical Association meeting in 1950, reported on 1,229 cases occurring in Los Angeles County in 1949. The results of his analysis indicated no clinical or statistical significant deviation between the actual and the expected incidence of poliomyelitis developing in Los Angeles County during 1949 in patients recently tonsillectomized even in the months of July through October. He concluded there was no reason found to discontinue adenotonsillectomies during the summer months.

Goerke[24] (personal communication) believes the rate of bulbar involvement of poliomyelitis cases is significantly higher following adenotonsillectomy than in cases not following these operations and is convinced that it is a great responsibility on the individual physician to perform an elective adenotonsillectomy during the summer months in Los Angeles. He feels that deferment of elective adenotonsillectomy is wise during the increased incidence of poliomyelitis, which in the city of Los Angeles is from June through November.

The Texas, 1950, poliomyelitis cases totaled 2,778 and the peak month was July with 571 cases. A total of 1,492 Epidemiological Report Forms were received by the Division on Epidemiology. Of this number, 42 answered yes in regard to recent (three weeks) tonsillectomy or dental operation, but this question was not answered on 28 of the forms. The bulbar cases totaled 91 and five of these were preceded by recent tonsillectomy or dental operation.

Greenberg[25] (personal communication) states that in his analysis of the 1949 epidemic in New York City the actual number of cases of poliomyelitis occurring within one month after operation was three and one-half times the expected number. Also, for the years 1944 to 1949 the percentage of cases with bulbar paralysis within one month of operation was 17 times greater than in a control group of nontonsillectomized children.

Aycock,[26] in 1942, reviewed 170 cases of poliomyelitis following tonsillectomy within 30 days and 121 (71 per cent) of these were either bulbar or bulbospinal in type. In 1949, Aycock presented to the Eastern Section of the American Laryngological Society a paper[27] which is worthwhile reading to obtain his well expressed attitude toward tonsillectomy and poliomyelitis, but a synopsis here would not do the paper justice. Some of Aycock's views have been incorporated in the following comment and conclusions.

COMMENT AND CONCLUSIONS.

There is unmistakable evidence that the attack rate of bulbar poliomyelitis in many epidemics has been found to be significantly higher in recently tonsillectomized patients, and, evidence is increasing that bulbar poliomyelitis rather than the spinal form of the disease is more likely to occur in patients tonsillectomized at any time than in those with tonsils intact.

This reviewer agrees with Aycock's statement that the fact which proves beyond a doubt that there is a relationship between tonsillectomy and poliomyelitis is the greater frequency with which bulbar poliomyelitis is seen to follow tonsillectomy by a specific interval. Other types of poliomyelitis do not follow tonsillectomy by this specific time interval.

The danger of bulbar poliomyelitis following tonsillectomy is almost zero except during periods of increased incidence or epidemics when, although the risk is not great, it is definite and real and increases in proportion to the increase in incidence. Since the hazard of the tonsillectomy-bulbar poliomyelitis sequence is small, it should not preclude adenotonsillectomy for definite medical indications, but at the same time it is advisable to take into consideration the timing of the operation, which is elective in most cases.

Adenotonsillectomies should not be performed during an epidemic or on patients with known recent exposure to poliomyelitis, and, the operation should be delayed when there is an increased incidence of poliomyelitis in the community unless postponement jeopardizes the patient's health to a greater degree.

The practice of the Boston hospitals appears logical. These hospitals follow a uniform procedure agreed upon by them and based on information and recommendations furnished by their State Board of Health relative to increase in prevalence of the disease in any of the various areas served by the hospitals.

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DIHYDROSTREPTOMYCIN-BORIC ACID POWDER IN THE TREATMENT OF AURAL DISCHARGE.

A CLINICAL REPORT.*†

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Notwithstanding the advances and modifications that have occurred in the practice of otolaryngology in the past 15 years, the problem of aural discharge remains one of the most common to confront the otologist in his daily office routine. During these years many new drugs have come to the treatment table, some to be discarded, others to find a permanent place in our armamentarium. The purpose of the present report is to record clinical experiences with one of the new antibiotics—dihydrostreptomycin—used in conjunction with an acid, drying agent—boric acid—and to present biochemical and bacteriological data that may offer some explanation of the beneficial results observed in the clinical use of this mixture.

REVIEW OF THE LITERATURE.

Comprehensive and repeated studies of the bacteriology of aural discharge have been reported in recent years and it does not seem necessary to include well established data; however, a review of the literature directly concerned with the present report seems pertinent.

Collins and Hughes[1] studied the bacterial flora occurring in 26 ears of 23, previously untreated patients with chronic

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otitis media, and they observed that Gram-negative bacilli were more frequently present than Gram-positive cocci. Repeated cultures of flora from the middle ear cavities of certain patients treated with local penicillin revealed a complete change from the original, predominantly Gram-positive organisms—sensitive to penicillin—to Gram-negative bacilli—resistant to penicillin. They concluded that, "The presence of the secondary organisms, such as the 'coliform' bacilli, B. proteus and diphtheroids was undoubtedly one of the complicating factors limiting the successful treatment of chronic suppurative otitis media by penicillin."

Pseudomonas aeruginosa was the predominant organism in 45 of 100 consecutive cases reported by Salvin and Lewis.[2] In another study[3] of 72 ears, the same organism was found in 67 per cent of the cases. Other Gram-negative bacilli were cultured in 17 per cent. These organisms were also noted in other cases of otitis externa, and the failure of local and parenteral penicillin therapy has been ascribed to their presence in the exudate.[4]

Holt and Snell[5] reported 20 cases of chronic otitis media in which cultures from 26 ears had been made. *Pseudomonas aeruginosa* was present in 12 instances and *Bacillus proteus* in 14 cultures.

Senturia[6] states, "In a recent report on 135 cases of external otitis observed at Randolph Field, Tex., and in reports from New Guinea and the Solomon Islands, it was shown that fungi were found in a small number of cases and that Gram-negative bacilli (especially the Pseudomonas group) were found in a very large percentage of cases observed in these tropical and subtropical areas."

Douglas,[7] in an analysis of the bacteriology of 142 ears, reported the prevailing organisms to be "Staphylococcus, Proteus, Pseudomonas and Streptococcus" in the order named.

Hayes and Hall,[8] in a clinical and bacteriological study of 160 cultures from 100 cases, reported that the resistant etiological agent in chronic suppurative otitis media and external otitis was predominantly *Pseudomonas aeruginosa.*

Gill[9] presented results of 252 cultural studies of the external ears of 168 patients and reported that bacteria were present in 117 cases, bacteria and fungi in 48, and fungi alone in three cases. Gram-negative bacilli occurred most frequently (104 cultures). Staphylococci were present in 96 cultures.

All of these authors have stressed the importance of using an agent that is effective against Gram-negative bacilli in the management of aural discharge.

The effect of vehicles on the antibacterial action of various substances was extensively studied by Senturia and Doubly.[10] Using the cultures of *Staphylococcus aureus* and *Pseudomonas aeruginosa,* they found the action of streptomycin to be unimpaired by such vehicles as distilled water, glycerin, Carbowax and "Carboprop." On the other hand, the effectiveness of streptomycin was completely inhibited when the vehicle used was lanolin, Aquaphor or Hydrosorb, all water-insoluble vehicles.

The susceptibility to infection may be due to such predisposing factors as moisture together with maceration of the skin, absence of cerumen, and the accumulation of dead, keratinized, horny tissue with the neutralization of the antibacterial, acid cloak.

The pH of the cerumen and of the skin of the canal, which is normally acid, is of special importance. In healthy ears, the pH is within the acid range. Fabricant and Perlstein[11] studied the pH of the external ear in 27 infants, 44 children and sixty adults with no apparent lesions in their external auditory canals, and found that pH values fell within the acid range in the majority of instances (pH range: 5.0 to 7.8). They conclude, "The management of the more difficult and more resistant cutaneous infections can be improved by a better understanding of the manner in which the skin resists and harbors bacteria and of some of the mechanisms by which bacteria produce harmful effects. Several factors play a rôle

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
1	Itching, deafness	Unknown	Diffuse otitis externa	5	Improved
2	Itching and crusted ears	5 years	Diffuse crusting	11	Allergic dermatitis
3	Discharging left ear	1 week	Diffuse purulent otitis externa	1	Not known
4	Painful and swollen left ear	several days	Unlocalized furuncle	13	Improved
5	Painful ear	several weeks	Diffuse otitis externa with bloody blebs	10	Improved
6	Discharging left ear	1½ years	Diffuse otitis externa	1	Not known
7	Itching and crusted ears	1 year	Diffuse otitis externa with crusting	4	Improved
8	Irritation and itching	several weeks	Mild otitis externa	1	Improved
9	Itching and discharging ears	2 months	Diffuse otitis externa with crusting	5	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
10	Painful, itching, and discharging ears	2 years	Severe bilateral diffuse otitis externa	4	Improved
11	Painful crusted ear and face	Unknown	Crusting dermatitis	1	Not known
12	Itching, crusted, discharging ears	4-6 months	Neuro-dermatitis?	9	Allergic dermatitis
13	Painful ears	6 months	Severe diffuse otitis externa	7	Improved
14	Swollen, painful, itching ears	2 days	Severe diffuse otitis externa	4	Improved
15	Itching, crusted, swollen right ear	1 week	Diffuse otitis externa with crusting	1	Not known
16	Crusted, weeping left ear	4 days	Diffuse otitis externa with crusting	2	Improved
17	Itching and discharging ears	6(7) months	Diffuse otitis externa	4	Improved
18	Itching, discharging ears	1 year	Diffuse otitis externa with crusting	1	Not known

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
19	Itching ears	6(?) months	Diffuse otitis externa with discharge	4	Improved
20	Itching, discharging ears	6(?) weeks	Diffuse otitis externa with discharge	2	Improved
21	Swelling and weeping ears	6 + months	Diffuse otitis externa	3	Improved
22	Itching and discharging ears	Unknown	Diffuse otitis externa with excoriation	1	Not known
23	Itching and stuffy ears	1 + weeks	Diffuse otitis externa with crusting	1	Not known
24	Crusting, both ears	2 years	Diffuse otitis externa with crusting	10	Improved
25	Crusted and itching ear	3 + months	Diffuse otitis externa with excoriation	15	Improved but marked allergic reaction to treatment

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
26	Itching and discharging ears	Unknown	Otitis externa with discharge	5	Improved
27	Crusted and itching ears	2+ years	Diffuse otitis externa with crusting	6	Improved
28	Swollen and painful ears	4+ days	Diffuse otitis externa with unlocalized furuncles	4	Improved
29	Itching and discharging ears	3 months	Diffuse otitis externa with crusting	3	Improved
30	Itching and crusting right ear	3+ months	Diffuse otitis externa with crusting	4	Improved (Allergic factor also)
31	Discharging and itching ears	3 months	Diffuse otitis externa with foul debris	2	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMEN	RESULTS
32	Stuffy and uncomfortable left ear	Many years	Old radical cavity	3	Improved
33	Painful and bleeding right ear	1 day	Traumatic laceration canal and drum	13	Complete healing of perforation
34	Injury left ear by tree limb	1 day	Abrasion canal wall perforation of drum	3	Improved, no infection
35	Discharging right ear	10 years	OMSCH, central perforation	5	Ear dry, apparent closing of perforation
36	Discharging right ear	2 weeks	Otitis media, large central perforation	3	Ear dry
37	Discharging right ear	From childhood	OMSCH, central perforation	3	Ear dry
38	Discharging left ear	64 months	Large double perforation, left drum	3	Improved (TB Patient)
39	Discharging right ear	9 days	OMSCH, central perforation	7	Ear dry
40	Painful, discharging left ear	15 years	Otitis externa & OMSCH, central perforation	6	Ear dry

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
41	Discharging left ear	Many years	OMSCH, marginal perforation	10	Ear dry
42	Fullness, irritation left ear	15 years	Left radical mastoid cavity	-	Improved
43	Pain, deafness left ear	2 weeks	OMSA, central perforation	6	Improved
44	Discharging left ear	Many years	OMSCH, marginal perforation, granulations	5	Ear dry
45	Discharging right ear	Many years	Radical mastoid cavity	-	Improved
46	Discharging left ear	Many years	Left radical mastoid cavity	-	Unimproved tubal discharge continues
47	Discharging right ear	3 months	OMSCH, central perforation -	18	Ear dry
48	Bloody discharge right ear	Many years	OMSCH, marginal perforation	-	Allergic reaction mastoid surgery advised

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
49	Discharge left ear	8 years	OMSCH, marginal perforation with granulations	7	Ear dry
50	Running right ear	6 months	OMSCH, marginal perforation	1 year	Unimproved patient did not return
51	Itching discharging right ear	Many years	Modified radical mastoid cavity	7	Ear dry
52	Irritated discharge left ear	13 years	Radical mastoid cavity	11	Improved but allergic reaction occurred
53	Discharge right ear	10 years	OMSCH, marginal perforation	8	Ear dry
54	Discharge left ear	many years	OMSCH, central perforation	4	Improved
55	Discharge right ear	10 days	OMSCH, central perforation	7	Ear dry
56	Discharging painful left ear	many years	OMSCH, central perforation	5	Improved

CASE NUMBER	COMPLAINTS	DURATION	DIAGNOSIS	NUMBER OF TREATMENTS	RESULTS
57	Discharge deafness left ear	many years	OMSCH, marginal perforation	8	Ear dry
58	Discharge deafness both ears	many years	Right OMSCH with cholesteatoma Left OMSCH, marginal perforation	5	Improved
59	Discharge right ear	3 days	OMSCH, central perforation. Left same	4	Ear dry
60	Discharge left ear	1 week	OMSCH, central perforation	4	Improved
61	Deafness right ear	1 week	Large perforation right drum, traumatic following blow	9	Drum healed and intact

in the remarkable protection afforded by the normal human skin against bacterial invasion. These factors, however, are imperfectly understood, and further basic study is necessary.

"A factor of considerable importance is the pH of the skin. The surface of the skin is enveloped by an 'acid mantle,' which exhibits pronounced defense against bacterial invasion. Determinations of the pH of the skin by other investigators reveal the following facts: *1.* The seborrheic areas of the body are more alkaline than other parts of the skin's surfaces; *2.* the alkaline areas are less bactericidal than the rest of the skin, thus being predisposed to cutaneous infection; *3.* in most cases of vesicular eczema there is a shift of the pH of the skin toward the alkaline side; *4.* the highest degree of alkalinity is present in fissures associated with eczematous processes, and *5.* moisture, dead horny tissue and relative alkalinity are four favorite conditions for the propagation of many forms of fungi."

In view of the foregoing cansiderations, it would seem that the ideal agent for the treatment of aural discharge should have the following properties: *1.* it should be strongly effective against Gram-negative organisms; *2.* the vehicle used with the agent must not impair its efficiency; *3.* the agent should be easy to apply and should not interfere with the elimination of discharge or debris; *4.* it should be in an acid medium, and *5.* it should have a drying effect.

Reports on the use of streptomycin and its modification, dihydrostreptomycin, have occurred with increasing frequency in the medical literature since 1947. Pulaski and Matthews[12] reported favorable results in five cases of chronic otitis externa, treated with a solution of streptomycin (5 micrograms per cubic centimeter). They also treated 13 patients with chronic otitis media and reported that 11 were benefited. They suggested that greater concentrations of the antibiotic agent might prove of further benefit.

Callaway[13] reported dramatic improvement of a case of severe infection of the ear by *Pseudomonas aeruginosa* that had proved resistant to many forms of treatment over a period of nearly one year. He used an aqueous solution of streptomycin containing 2,500 units per cubic centimeter.

Lundon[14] treated a series of 20 outpatients of the Queen Mary Veterans' Hospital in Montreal, Canada, all of whom had chronic suppurative otitis media. The duration of treatment varied from one to 12 weeks. Of this group, 15 responded favorably, "as evidenced by 'dry ears' and absence of pathogenic bacteria"; five cases were regarded as therapeutic failures.

Miller *et al.*[15] used dihydrostreptomycin ointment, 5 micrograms per gram, in the treatment of 124 cases of pyogenic infections of the skin. Nine of these were diagnosed as otitis externa. These authors demonstrated that formulas of oil in water and greaseless base yielded a superior release of the antibiotic. They also emphasized the fact that good results with dihydrostreptomycin usually occurred rapidly, but that prolonged use of the agent was not advocated, since poor results might be due to drug-fastness. The treatment caused a vesicular dermatitis in four patients of the series (3.7 per cent).

Harris *et al.*[16] presented bacteriological and clinical observations made in 27 ears "with suppurative otitis, under treatment with local instillations of streptomycin solutions. Clinical improvement occurred in about half these ears. Drainage stopped during the treatment and did not recur during a follow-up period of from two to six months." They used streptomycin in sterile, isotonic sodium chloride solution, with concentrations varying from 1,000 to 25,000 units per cubic centimeter. They noted that resistance of organisms, previously sensitive to streptomycin, appeared during the course of treatment in a large number of cases, despite the fact that the concentration of streptomycin instilled was at least 20 times greater than the minimum concentration required to inhibit the growth of the organisms *in vitro*, by the usual sensitivity tests, before treatment was started.

Senturia and Broh-Kahn[17] treated a series of 54 consecutive cases of diffuse otitis with streptomycin, incorporated in "Carboprop" [60 parts of Carbowax (4,000) and 40 parts of propylene glycol]. Favorable results were obtained when a concentration of 5 mg. of streptomycin per gram was used. When concentrations of 1 mg. of streptomycin per gram, or less, were used the results were either poor or inconclusive. Controls treated with "Carboprop" alone did not produce a comparable improvement.

CLINICAL AND LABORATORY INVESTIGATION.

To date, all available reports dealing with the topical use of streptomycin have recorded the use of this antibiotic in an aqueous solution or in some form of ointment or similar medium. In May, 1949, following the conclusion of a scientific session at a national meeting, a group of otologists were informally discussing the treatment of various conditions. At that time, someone suggested the use of streptomycin incorporated in boric acid powder. In view of the fact that a drying agent—such as boric acid—has always been one of the most successful in the author's experience, he decided to begin an extended clinical trial of such a method of therapy.

The method of treatment of aural discharge, devised for this clinical study, is as follows: *1.* the canal is carefully cleaned of all debris, using 70 per cent ethyl alcohol; it is then dried; *2.* dihydrostreptomycin-boric acid powder (one part of the former, three parts of the latter) is insufflated generously into the canal, using a DeVilbiss powder blower; *3.* the patient is instructed to leave the ear untouched until the following morning, and at that time he is to start the use of five drops of boric acid-alcohol and is to continue this three times daily until the next office visit. Office visits were spaced at two or three times a week whenever possible, but this schedule does not hold for all the patients included in this report. Throughout the years, boric acid-alcohol drops have remained a standby with many otologists. The formula may vary slightly among otologists, but the prescription given the patients in the present report is as follows: Ethyl alcohol, 70 per cent; boric acid powder, quantity sufficient to saturate.

In view of the relative importance of maintaining the acidity of the skin, the pH of the substances used in this clinical investigation was determined* with the following results:

Two hundred mg. of dihydrostreptomycin-boric acid powder der mixture, dissolved in 5 ml. distilled water (practically all of which when into solution)—pH 4.32.

Boric acid powder, U. S. P.—pH 4.05.

Boric acid-solution—pH 5.50.

Boric acid-alcohol solution, diluted one-half with distilled water— pH 5.50.

These values demonstrate that both of the agents used in this study assist in maintaining the acid condition of normal skin, and it may be that the major value of boric acid-alcohol drops can be attributed to this factor.

BACTERIOLOGICAL STUDIES.

Pure cultures of laboratory strains of *Pseudomonas aeruginosa* and *Staphylococcus aureus* were streaked on agar plates and were then treated with dihydrostreptomycin-boric acid powder and with boric-alcohol solution. In the former culture plates, wide areas of bacterial inhibition were clearly visible, but in the latter instance, bacteriostatic action was extremely limited. Fig. 1 demonstrated the bacteriological results of typical laboratory studies made with the agents described and employed in this clinical study.

The observed *in vitro* action of dihydrostreptomycin alone was in accord with reports of other investigators, but nothing in the literature has suggested the enhanced bacteriostatic action of the dihydrostreptomycin-boric acid powder mixture.

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*All pH determinations were made using the Beckman Glass Electrode pH Meter (Model G), with solutions at a room temperature of 23° Centigrade.

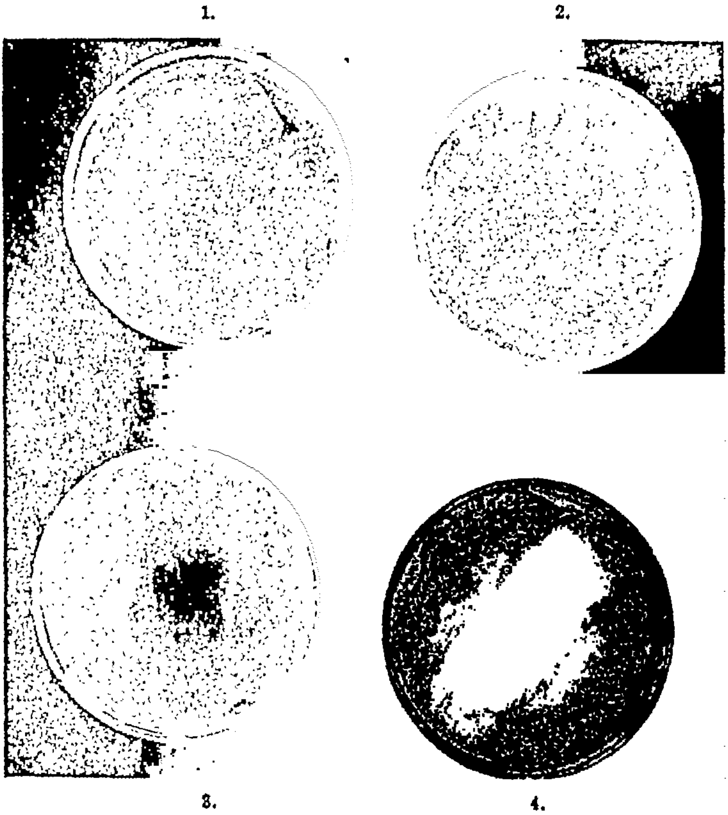

Fig. 1. Pus collected on a swab from a patient with chronic mastoiditis was suspended in 1 ml. of infusion broth. One drop of the suspension was placed in the center of each of four extract-agar and four blood-agar plates and spread over the surface of the plates with a glass rod. Ordinary streak plates of the suspension were made at the same time on extract agar and blood agar.

One extract and one blood-agar plate were left as controls. A loopful of powdered boric acid was streaked across the center of one extract and one blood-agar plate; a loopful of powdered dihydrostreptomycin was streaked across another similar set; and dihydrostreptomycin-boric acid powder mixture was streaked across a third set. All of the plates were incubated for 24 hours.

The illustration shows the appearance of the extract-agar plates after incubation.

Plate 1. Control.

Plate 2. With powdered boric acid.

Plate 3. With powdered dihydrostreptomycin.

Plate 4. With dihydrostreptomycin-boric acid powder mixture.

The streak plates contained colonies of the following species: Pseudomonas aeruginosa, Aerobacter aerogenes, hemolytic Staphylococcus aureus, Streptococcus fecalis, and a diphtheroid which was not completely identified. The colonies appearing in the area of inhibition on Plate 3 were the diphtheroid and Streptococcus fecalis.

It has been pointed out that streptomycin is more effective when used in alkaline medium.[19] While this may be true when used in dilute solutions, the present studies suggest that it is also strongly effective in high concentrations, even though incorporated in markedly acid media.

CLINICAL REPORTS.

Case histories taken from a private practice seldom are ideal or satisfactory for statistical analysis. Controls usually cannot be used, and the factors of time, economical situation of the patients, and the difficulty of obtaining follow-up data in many instances often result in incomplete analyses. Patients who receive substantial benefit from the therapy used frequently fail to return for a check-up when their major complaints are relieved. This results in incomplete reports of cure and recurrence of symptoms.

Since bacteriological data in these presently reported cases were in general well established, bacteriological cultures were not made routinely, but in those cases in which this was done, the findings were in accord with those given in the literature as herein reviewed.

The cases observed present a variety of conditions that do not lend themselves to a tabular chart report; therefore, each case has been abstracted as briefly as possible, and all findings not directly related to the chief complaint have been omitted. For the purpose of simplification, the dihydrostreptomycin-boric acid powder, which was used for treatment in this series of patients, has been named "strepto B," and the boric acid-alcohol drops have been designated as "B and A drops." These terms are used throughout the case reports.

Otitis Externa.—It is generally accepted that factors other than bacterial infection may play a part in many cases of *otitis* externa. Allergy and other constitutional disorders must not be overlooked as etiological agents. When, however, the discharge is reduced or stopped after two or three treatments with an antibacterial agent, it seems reasonable to assume that in these cases infection is the major factor.

It has been found difficult to fit the following cases into any formal classification and, therefore, several descriptive terms have been used in specific instances instead. Some authors have used such terms as "suppurative" and "eczematoid" for categorical, differentiation purposes, but these terms are not generally understood or accepted, and so are not used here.

SUMMARY AND CONCLUSIONS.

1. Dihydrostreptomycin-boric acid powder appears to be an effective agent in the treatment of aural discharge.

2. The acidity of this preparation has been determined. Evidence taken from the literature shows that acidity is desirable in the treatment of aural discharge.

3. Bacteriological evidence from the present study suggests that this mixture is more effective than is dihydrostreptomycin alone.

4. Moisture, which encourages bacterial growth, is reduced by the drying effect of this treatment.

5. Vesicular skin lesions occurred in a small percentage of cases when this treatment was used, but they cleared rapidly upon cessation of the application.

6. Thirty-one cases of otitis externa were treated with dihydrostreptomycin-boric acid powder. In 21 of these cases improvement was obtained. In seven cases results were unsatisfactory or inconclusive. In three cases vesicular dermatitis occurred.

7. Thirty cases of otitis media, mastoiditis or tympanic trauma were treated with dihydrostreptomycin-boric acid powder. In 22 of these cases improvement was noted. In six cases results were unsatisfactory or inconclusive. In two cases vesicular dermatitis occurred.

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BOOK REVIEW.

Atlas der Hals- Nasen- Ohren-Krankheiten. By C. v. Eicken and A. Schulz van Treeck. Third Edition. 199 pages with 470 illustrations, many of them in color. Stuttgart: Georg Thieme, Verlag. 1951. Price DM 78. American agents, New York: Grune & Stratton, Inc.

The reproduction of colored drawings and photographs in this atlas is beautiful, although in many instances the coloring is too vivid. The detailed work in most of these drawings is highly commendable.

There are many conditions shown which, in our present day therapy, in this country, are not seen in the advanced stages illustrated. One has the feeling, after turning through the pages, that the atlas is not as up to date as it should be. Many of the descriptions accompanying the illustrations are too brief, and yet ample space is present on many pages.

Such an atlas should be of value to any student beginning a study of diseases of the ear, nose and throat. W. P. C.

THE GRADUATE SCHOOL OF MEDICINE OF THE UNIVERSITY OF FLORIDA.

The sixth annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held Jan. 14-19, 1952. As in previous years, Miami Beach will be the host city.

The lectures on ophthalmology will be presented on Jan. 14, 15 and 16; those on otolaryngology on Jan. 17, 18 and 19. Lecturers for the courses in ophthalmology will include such distinguished teachers as Dr. Alson E. Braley, Iowa City, Iowa; Dr. John M. McLean, New York City; Dr. Wendell Hughes, Hempstead, Long Island, New York; Dr. Irving H. Leopold, Philadelphia, Pa., and Dr. Bruce Fralcik, Ann Arbor, Mich.

Outstanding otolaryngologists who will lecture include Dr. Fred W. Dixon, Cleveland, Ohio; Dr. Thomas D. Galloway, Evanston, Ill.; Dr. Anderson G. Hilding, Duluth, Minn.; Dr. Moses H. Lurie, Boston, Mass., and Dr. Henry L. Williams, Rochester, Minn.

The Southern Section Meeting of the American Laryngological, Rhinological and Otological Society will be held Monday, Jan. 14, at Atlanta, Ga. The Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology planned its meeting in Havana the week following our seminar. Registrants of the seminar might like to include one or both these other meetings in their southern trip.

All meetings will be held at the Sans Souci Hotel. Registration fee for the seminar is $40.00. A check for $10.00, payable to the University of Florida Midwinter Seminar, must accompany your application. This is not returnable. The remainder of the registration fee will be paid at the seminar desk at the Sans Souci Hotel on your arrival.

Convention rates are good for the seminar and for any period before Jan. 14, but after Jan. 19 the regular, higher rates are effective.

Division of Ophthalmology: Shaler Richardson, M.D., Jacksonville; Nelson M. Black, M.D., Miami; Charles Boyd, M.D., Jacksonville; Bascom Palmer, M.D., Miami.

Division of Otolaryngology: Marshall Taylor, M.D., Jacksonville; Walter T. Hotchkiss, M.D., Miami Beach.

MISSISSIPPI VALLEY MEDICAL SOCIETY
1952 ESSAY CONTEST.

The Twelfth Annual Essay Contest of the Mississippi Valley Medical Society will be held in 1952. The Society will

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

December 1, 1951.

Audicon Models 400 and 415.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Audivox Model Super 67.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1.
Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer Mears Radio Hearing Device Corp., 1 W 34th St., New York, N Y

Micronic Model 303; Micronic Star Model

Manufacturer Micronic Co 727 Atlantic Ave Boston 11, Mass

Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model 45.

Manufacturer Microtone Co, Ford Parkway on the Mississippi, St. Paul Minn Minneapolis 9 Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer National Hearing Aid Laboratories 815 S Hill St., Los Angeles 14, Calif

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4, Otarion Models F-1, F-2 and F-3; Otarion Model G-2

Manufacturer Otarion Hearing Aids 159 N Dearborn St. Chicago Ill

Paravox Model J (Tiny-Myte); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Manufacturer Paravox Inc 2056 E 4th St., Cleveland Ohio

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer E. A Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa

Rochester Model R-1; Rochester Model R-2.

Manufacturer Rochester Acoustical Laboratories Inc 117 Fourth St S W Rochester, Minn

Silvertone Model 103BM.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model J-92.

Manufacturer: Sears-Roebuck Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, Minneapolis 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube Model No. 300.

Manufacturer: Trimm, Inc., 400 Lake St. (P. O. Box 489), Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.

Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal.

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Sect.: Dr. Pedro Hernandez Gonzalo, Calle 8, 358, Havana, Cuba.
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Time and Place: Havana, Cuba, January 20-24, 1952.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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